BRAIN.

PARTS II. and III., 1895.

Original Articles and Clinical Cases.

BRAIN ORIGIN.[1]

BY SIR WILLIAM BROADBENT.

THE honour of presiding over this Society is one of which I am peculiarly sensible, and I am grateful to the Council and Members for bearing in mind that at one period of my career I was more or less a neurologist, though the name was not then invented. I was, in fact, one of a band of men enthusiastically engaged in the study of the nervous system and the diseases to which it is liable, and my oldest and best friends are still among those with whom I thus became associated.

I might well take as the subject of my address the stupendous progress realised in our knowledge of the physiology and pathology of the nervous system since that time. Lockhart Clarke was only then giving us a definite idea of the structure of the spinal cord and bulb, and Brown-Séquard was publishing his brilliant researches on the function of its different columns and strands of fibres, and on the motor and sensory paths. The ophthalmoscope, Helmholtz's splendid invention, had not then been applied to the study of diseases of the brain. The two last names, both immortal in the annals of science, I bring together because they have been erased by death from the list of corresponding members of this society during the past year. At that time Charcot,

[1] Being the Presidential Address to the Neurological Society of London, January, 1895.

whose loss we had to deplore in the previous year, had only just entered upon the extensive field of work afforded by the Salpêtrière, and his gigantic contributions to neurology were still in the womb of the future. Virchow's teachings with regard to embolism and its results had not been applied to cerebral pathology, and our teachers of that day spoke of red or inflammatory softening. The significance of the knee-jerk was unknown. The localisation of cerebral functions was scarcely even dreamt of. Broca's identification of the third left frontal convolution as the seat of articulate speech, the first instance of the kind, had, it is true, been established for some time as an isolated fact. Then as Dr. Hughlings Jackson was pointing the way by a new interpretation of clinical observations, Hitzig and Ferrier stepped in, and, the latter especially, by a series of experimental investigations, as profound in conception and as fruitful in result as any in the history of physiology, mapped out the cortex according to the functions of the different convolutions.

In the short time during which I shall occupy the attention of the Society, however, I shall not attempt any such comprehensive review of the progress in our knowledge of diseases of the nervous system or an exposition of any special form of nerve disorder. Still less shall I emulate former Presidents of this Society who have made of their introductory addresses important contributions to our knowledge of the anatomical structure, or of the physiology of the nervous system. I should only lay myself open to criticism from the youngest member here present, but I am encouraged by the example of Dr. Gowers to carry out the idea which suggested itself to me as soon as I learnt that the responsibility of addressing you had devolved upon me. It is to bring to your notice certain speculations as to the nature of nerve force and as to the constitution and mode of action of the nervous system which have long occupied my mind.

Dr. Gowers, in his admirable address before the Medical Society of Manchester, has rendered me a double service. He has vindicated in eloquent and convincing language the utility of speculation and hypothesis as inspiring and

directing thought, and suggesting and guiding experiment, thus clearing my way and making any such effort on my part unnecessary, and again by his lucid and exhaustive discussion of the dynamics of life, he has spared you and me much detailed exposition, which in my hands might have been tedious. He has given increased vividness and precision to the notion of nerve force as a mode of motion correlated with the more familiar modes of motion, heat, light, electricity, chemical action, and mechanical energy, and also to our ideas as to the nature of the various stimuli which excite nerve action, pointing out that, like the concussion which explodes nitro-glycerine, or the heat which fires gunpowder, or the electric spark which determines the sudden combination of a mixture of oxygen and hydrogen, they are essentially added motion.

Nerve action, whatever the difficulties of giving a chemical explanation, can scarcely be other than motion liberated by molecular change, in other words by chemical change in the highly specialised nervous structures.

We cannot so clearly as in the case of muscular action show that the development of nerve force is attended with an increased output of CO_2 and water and obtain a quantitative evaluation of it by the amount of oxidation required for its production, but I think we may say that we have absolute proof that nerve force is liberated by a process which for want of a better term we must call chemical in the dependence of the functional activity of the great nerve centres upon a due supply of oxygen. This is best shown by the successive suspension from above downwards of the functions of the cerebral cortex, basal ganglia, and of the vital bulbar reflexes when the supply of the oxygen is cut off, or when the oxygen of the hæmoglobin is replaced by the anæsthetic gases or vapours. From the fact that oxygen so evidently takes part in the process, it would almost seem as if the nerve motion were liberated by atomic rather than molecular changes, though this is not a necessary conclusion.

There are many considerations which appear to indicate that nerve force must be characterised by intensity rather

than by quantity—that, for example, it would compare in quantivalence of motion with static or faradic electricity rather than with galvanism. A single cell with an adequate coil and core can yield faradic currents of intolerable strength, and it has been said that the electric force required to dissociate the hydrogen and oxygen of a grain of water would make a very respectable thunderstorm.

Among these considerations is the fact already alluded to, that it has never been possible to distinguish and measure the products of the oxidation by means of which nerve energy is liberated. Another is the remarkable promptitude of nervous operations of every kind, and the accurate adjustment of response to stimulus. Yet another is the marvellously minute dose of drugs acting upon the nervous system which is sufficient to produce striking effects. If the correlation of forces is to apply to the physiological effects of drugs—which is an article of scientific faith—then the chemical change in the morphia molecule must be equal and opposite to the change in the constituent molecules of the cerebral cortex which the morphia neutralises when it suspends the cortical functions.

Now the chemical correlative of intensity as the characteristic of nervous energy is the existence in the nerve centres of a substance possessing high chemical tension. By this is meant, not simple instability, not necessarily instability at all, but a molecular structure in which the atoms are grouped in a way which is not in accordance with what may be called their natural affinities, so that if once free to move they would rearrange themselves more or less abruptly and perhaps violently in a totally different manner.

Instability may be described as a readiness to yield to the disintegrating influences of forces acting from without; chemical tension implies the existence of an intrinsic tendency which under special conditions determines change in a definite direction and with a certain degree of energy. Nitro-glycerine, for example, or gun-cotton does not undergo gradual decomposition, and the former even resists the application of flame, while concussion determines in both a re-arrangement of the constituent elements attended with violent explosion.

Now nitrogen is the element which lends itself to sudden disruption of a complex molecule. It has very weak affinity for hydrogen and oxygen, none at all for carbon, while carbon, hydrogen and oxygen have powerful affinities for each other. The tension in nitro-glycerine and gun-cotton is brought about by the substitution of NO_2 for hydrogen in the molecule of glycerine or cellulose. Before this the molecule was stable but was ready to burn; *i.e.*, its carbon and hydrogen to leave their combination with each other and combine with oxygen. The introduction of additional oxygen in the NO_2 upon which the associated nitrogen has only a feeble hold may be compared to the mixing of oxygen with coal gas.

The chemical tension by means of which nerve force is evolved would not be of the same character as that exemplified by nitro-glycerine but would more probably resemble that produced by the forced departure from a stable molecular type, as, for example, by the successive removal of hydrogen from the ammonium molecule. Amides, imides and nitrites are formed as one, two and three equivalents of hydrogen are abstracted, in which the affinity of nitrogen for hydrogen, which is satisfied in the ammonium molecule, is left increasingly unsatisfied, each step in the process, generating an increasing tendency to return to the natural molecular structure. It is this inherent tendency towards a normal balance of affinities and forces which for want of a better term we call chemical tension.

When we contemplate nervous force from a chemical point of view an obvious conjecture is that, whereas most forms of nerve action are attended with chemical change in the direction of oxidation and disintegration, the impinging of sensory impressions on the cerebral cortex, not giving rise to immediate response of any kind but registered as memory, may be attended with change of a converse character, integration of molecules and structure by means of which the mental picture is rendered more or less permanent. Another reflection which forces itself upon the mind is that, when the energy liberated in all the various forms of nervous activity has been accounted for by chemical changes in the

nervous structures concerned, and nerve force has been correlated with other modes of motion, we are left with sensation, consciousness and the higher mental operations as surplus. We cannot bring them into the reckoning at all; the phenomena of mind are outside and above and in excess of the material changes which subserve their manifestation.

Turning now to a speculation of another kind, I think I am not misrepresenting the prevailing idea as to the nature of nerve force, and as to the relation between the nerve centres themselves and between them and the periphery, when I say that nerve force is considered to be energy which is transmitted from point to point of the nervo-muscular apparatus; that, for example, in the production of muscular action a certain force is supposed to be generated in the centre and transmitted to the muscles upon which it acts as a stimulus, the muscles awaiting passively the added motion in response to which they develop mechanical energy. This idea of the origination and transmission of energy pervades our entire conception of nervous operations, a centre or end organ being looked upon as the passive recipient of an impulse or force which is evolved in the centre in which the energy is liberated and from which the impulse starts.

Now it has long seemed to me that a conception which better fits in with all the phenomena of nervous action is, that instead of this passive attitude of the periphery and centres one to the other and of the centres, sensory and motor alike, towards each other, the relation in all cases is one of mutual tension or resistance—that is, that at all points specialised for the liberation of nerve motion, latent energy is generated and exists in a potential form, which, in the absence of restraining conditions would become active, and that the restraining condition is a counter-balancing energy developed in the centres or peripheral nerve expansions with which any given point is connected by nerve fibres.

In a galvanic battery, which may be taken as an illustration, the powerful affinities of sulphuric acid and zinc are held in check so long as there is no connection between the carbon and zinc elements; and a state of tension or charge

exists in the wires carried from the carbon and zinc respectively which, when the number of cells is sufficiently great, is betrayed by the leap of a spark from one electrode to the other, or by the passage of a current through water which it decomposes on its way.

The telegraph wires, again, in the intervals during which no messages are passing are not simply in a passive state; they are charged with electricity by the batteries at the two ends which are held in an equilibrium of mutual resistance through the charge which they communicate to the wires, so that disturbance at one end in a plus or minus direction gives instant rise to a disturbance at the other, corresponding exactly both in direction and in degree. The telegraphic instruments are, so to speak, in touch with each other. The same may be said of the nerve centres and peripheral nerve endings; they feel each other by the exercise of a sort of opposing pressure from each end through the connecting nerves, like two fencers whose swords are crossed.

Without saying that this conception removes the difficulty in understanding the marvellous correspondence of sensations with impressions, or the equally marvellous adjustment of muscular movement to motor nerve impulses, it may perhaps bring it nearer to our comprehension.

All who have attempted to grasp the mystery of nervous action have recognised that the nerve endings are highly specialised and endowed with some powers allied to those of the centres. This is especially evident in the case of the sensory nerves, and attains its highest development in the retina and auditory nerve. The termini of the cutaneous nerves must equally be specialised though the apparatus is less conspicuous, and, just as it is not light or sound which is transmitted to the brain, but nerve motion set up in the retina or auditory expansions by luminous vibrations or sonorous pulsations, so nerve motion is excited in the nerve endings of the skin by impressions of various kinds. In the eye and ear it is seen that structures are employed similar to those found in the nerve centres, having presumably therefore similar endowments, and in particular the power of generating nerve energy, and although nerve cells are not

recognisable in the skin, the specialised nerve endings must possess a power corresponding to that of the retinal expansion. There can be no difficulty, then, in supposing that at the sensory terminal distribution generally, force is continuously generated and maintained at a certain degree of tension, which is balanced and resisted in the nerve centres by force there generated.

In the same way the relation between the motor nerve endings and the motor nerve nuclei in the cord would be one of balanced tension. The muscular tonus which gives rise to the knee jerk would appear to be the peripheral expression of such a relation.

The various centres again in functional association with each other will stand in this relation of mutual stress, each generating force which exactly balances that of the associated centre or centres during repose, and the one responding to the slightest change set up by a stimulus in the other.

We may compare this relation of different nerve centres to two groups of men engaged in a tug of war. There is no movement in one direction or another so long as the two sides are exactly balanced in strength, but let one man slip, or become exhausted, or another gain a better foothold, and there is at once a movement in one direction or another of every member of his own group, which is accompanied by an exactly corresponding movement in the other. Another illustration may be suggested as perhaps illustrating the relation between a centre and a nerve end—a small group of men at the long arm of a lever resisting or compelling movement of a much larger group at the short end.

This relation of mutual tension and reciprocal control covers the whole field of operations governed by the nervous system. No separate mechanism of centres and nerves is required for thermal, trophic and inhibitory operations.

The centres and nerves which subserve sensation and motion are also employed in the liberation and control of heat. The thermal centres supposed to be identified and localised by experiment practically coincide with and are the same as the motor centres. As regards the part taken

by the nervous system in the production and regulation of the body heat, it would appear from the occurrence of hyperpyrexia after fatal injuries to the brain, and in the course of certain diseases simultaneously with extreme nervous prostration, and from the rise of temperature sometimes observed after sudden death or very rapidly fatal disease, that the tendency in the blood and tissues is to oxidation and excessive heat production, this being held in control by the nervous system. The higher temperature found in the paralysed side in the hemiplegia points to the same conclusion. It seems probable that pyrexia may result sometimes from acceleration of oxidation in the structures overcoming the central thermotaxic control, at others from impairment of the control exercised by the nervous system.

In the same way this conception of the relation between the periphery and centres and between different centres goes far to explain inhibition. The normal relation between all parts of the nervous system would, in fact, on this hypothesis be one of mutual inhibition or control, and it would follow that a powerful impression on one centre, in other words, an unusual development of energy, at a particular point, might at the same time excite increased activity of one set of associated centres, and resist and annul increased motion in another centre or set of centres.

One of the most remarkable examples of inhibition is seen in the prevention of an attack of Jacksonian epilepsy by tightening a ligature round the limb which is the seat of the initial convulsion. A coarse lesion exists in some part of the motor cortical area; irritation dating from this lesion sets up from time to time convulsive movements in the limb whose motor centre is the seat of the disease, and yet although there is irritation in the motor centre itself, the convulsion can not unfrequently be arrested by a tight cord round the limb. A fact like this can scarcely be understood except on the hypothesis that the periphery and centres hold each other in mutual control.

So with regard to trophic influence. The nerve endings in skin and muscles are identified in their nutrition with the structures to which they are distributed. The muscles,

indeed, may almost be looked upon as end-organs of the motor nerves. Through this community of structure and nutrition with the nerve terminations it seems to me that the tissues will take part in the maintenance of the tension between periphery and centres, while in turn the nerves will influence the nutritional processes in the tissues just as, by way of analogy, heat and electricity condition chemical action.

We see nutrition of the tissues disturbed by influences transmitted both along motor and sensory nerves with or against the direction of the physiological nerve impulses. The results are more striking in the latter case, as is exemplified by acute bed-sores, herpes and ulcerations, all of which are excited by disturbing influences transmitted from the cord along the sensory nerves to the skin.

It is remarkable, again, that in herpes the most common if not the most interesting illustration of trophic lesions, the vesicles come out in scattered patches so that only a small part of the area of skin, to which the nerves conveying the disturbing influence are distributed, is affected. In some cases of frontal herpes, however, the skin of the entire region may subsequently undergo atrophy, and be left thin and glistening; and after a severe blow on the side I have seen two intercostal spaces crowded with vesicles from the seat of the injury to near the edge of the sternum. Perhaps Dr. Head, who has made such excellent use of herpes in his instructive researches with regard to cutaneous tenderness in visceral disease, can tell us whether the groups of vesicles occupy approximately the same situation in different cases. A certain degree of constancy is suggested by the patch which always appears on the back in zoster.

Herpes is often attributed to irritation or inflammation of a nerve, but it appears to be the result of a central disturbance, probably reflex, and not in any true sense of a neuritis. Labial herpes is curiously associated with the general commotion of the nervous system, of which rigor is the expression, and zoster in all its varieties is probably a visceral reflex. As Dr. Head has pointed out, the distribution of the patches is in relation with a segment of the cord,

and not with the termination of a given nerve or nerves. The neuritis which undoubtedly exists and keeps up the pain may be secondary to the central explosion, and, like the herpes, due to it.

Another point, of no importance in itself, but worthy perhaps of a few minutes' thought and attention, is the localisation of tactual impressions. From the earliest days of experimental investigation it has been recognised that division of a sensory nerve did not extinguish sensation in the skin to which it is distributed in the same definite way that division of a motor nerve paralyses muscles, and it is found that there is overlapping of sensory areas. This appears to prove conclusively that the localisation of an impression on the surface is not due to conduction of the nerve motion along an individual fibril starting from that particular point. Every papilla of the skin would, on this supposition, need to have two or more nerve fibrillæ, and it is already difficult to imagine that the cutaneous nerves can contain a sufficient number of fibrils to supply every point on the surface, however finely the axis bands may be sub-divided. The localisation of a sensation, it seems to me, must be effected by a sort of triangulation. In land surveying the exact position of a distant point is fixed by measuring the angles at the base of a triangle of which it forms the apex. So an impression made on a given spot excites nerve motion which is conveyed to the spinal cord by two or more nerve filaments approaching the spot from different directions, and its precise localisation is arrived at by continuous experience based upon the relative intensity of the motion set up in these converging nerve filaments. Impressions on adjacent parts will send currents a little more intense along one channel, a little less along another; and it must be by combination and comparison of the motion reaching the cord by these different channels that the point of origin is recognised. The entire papillary layer of the skin is, in my apprehension, a sentient expansion—a tactile retina, tapped at points more or less close by the sensory nerves.

A further difficulty in accepting the idea that the localisation of a sensory impression is due to transmission along a

single infinitely minute fibrilla proceeding directly from the point of origin to the cord, arises when we consider what happens when a sensory nerve is re-united after division. Nothing could be more improbable than that the coaptation should be so exact that every one of these fibrils should meet so that the continuity of each should be restored, without which sensory impressions would be wrongly placed. On the supposition under consideration the accurate localisation of sensations would not depend on the continuity of individual fibrils, but would be re-acquired by experience *pari passu* with the re-opening of the paths for centripetal impressions.

The reunion of a mixed nerve opens up further questions. It must happen that sensory fibres on the proximal side of the section will find themselves end to end with motor fibres on the distal side, and, in the absence of some condition which prevents it, they would unite, to the confusion of the nervous arrangements of the muscles and skin supplied. Apparently, however, this does not take place, and it must be assumed that efferent impulses, on the one hand, and afferent tendencies on the other, determine the establishment of the new communications. This implies some sort of dynamic influence from both proximal and distal ends, which is not easily understood on any other hypothesis than that advanced this evening, namely, that the nerve nuclei in the cord and nerve expansions in the muscle and skin stand in a relation of mutual tension, which tension exists in the nerves.

I will conclude this halting address by a few remarks on the much-debated question of the unity or multiplicity of the conductors of sensory impressions.

An important point which Dr. Gowers brings out is that the conduction of impulses along nerve fibres is effected by molecular change, as well as the evolution of nervous energy in the centres, and by change probably similar in kind, though differing in degree and especially in intensity. This conception appears to me to remove all necessity for postulating the existence of special conductors and centres for tactile, painful and thermal sensations.

The basis for this assumption is the fact that the

different forms of sensibility are not necessarily impaired concurrently, but that one may survive when the others are lost or perverted. But the degree or kind of motion making the impression is different in each case, and the motion set up in the conducting fibres and communicated to the sentient centres must be different. A tactile impression has only to be intensified to produce pain, and in certain states of the nervous system, central or peripheral, the lightest touch is intensely painful. Heat, again, and cold—which last, by the way, must subtract motion—carried beyond a certain point cease to be distinguished one from the other, the only sensation being one of pain.

Neither the experiments nor the clinical observations which have been held to demonstrate the separate paths and centres for these different modes of sensibility have at any time seemed to me conclusive; the lesions or morbid changes or drugs supposed to affect certain special fibres and centres may more probably impair the susceptibility of common or general conducting paths or termini to certain modes of motion, or their power of taking on such motion.

A consideration which has always seemed to me inconsistent with the existence of separate paths and centres for sensations of touch, pain and temperature is that they are so commonly lost in the same order, tactile sensibility surviving pain and the appreciation of heat and cold. The loss or impairment of sensibility to pain and variations of temperature, while tactile sensibility remains little affected, is stated to be so constant in syringo-myelitis as to serve as a diagnostic characteristic. Surely the structures of the cord are not affected in so constant an order that the tactile paths are never involved, while the fibres or networks which serve as the channels for pain and sensations of heat or cold are always destroyed.

Such a condition as the following, of which a well-marked example is under observation at the present moment, appears to me to be incapable of explanation by the hypothesis of multiple distinct sensory channels and centres, and to point to modification of a conducting and receptive apparatus common to all the different forms of cutaneous sensibility.

The condition is that tactile sensibility being good and sensitiveness to pain much exalted, there is absolute loss of sensibility to thermal impressions, any departure from an average temperature being felt as pain, whether in the direction of heat or cold, the patient being unable to say which of the two is being applied. The case is one of left hemiplegia, established three years ago during an attack of influenza. There was absolute loss of sensation over the entire left half of the body, and of motor power in the limbs. A considerable degree of movement has been recovered and improvement is still going on; there is no rigidity of the kind common after paralysis, but a certain degree of contraction of the flexor muscles still persists. Sensation slowly returned, pain being a marked feature from an early period.

If we consider the order of disappearance of the different forms of sensibility from the point of view that the peripheral receptive nerve expansions, the afferent fibres, and the central sensory termini are the same for all, this order becomes significant when compared with the order in which sensibility to different kinds of impressions must have been evolved. Tactile sensibility has been looked upon as the most intellectual of the cutaneous sensations, and the inference has been drawn that it was therefore the highest and latest in order of evolution, and consequently, according to the law on which Hughlings Jackson so constantly insists, it should be the first to disappear. But is this inference legitimate?

When we consider the rudimentary forms of life, it is clear that the existence of the creature must have depended upon an accurate response to surface impressions, which implies that such impressions must have been exactly localised, and that their character, *i.e.*, whether nutrient or not, must have been recognised. This is nothing more or less than a rudimentary sense of touch. The sense of pain would correspond to the recognition of impressions as being too violent for the safety of the part of the animal exposed to them. A localised recognition of temperature changes would not be required in the lower forms of animal life leading an aqueous

existence. We have thus as much conformity with the law that the order of dissolution corresponds with the order of evolution, as is met with in clinical experience generally.

I trust that these fragmentary speculations may not have been altogether unworthy of the attention of the Society.

POINTS RELATING TO THE WEBER-FECHNER LAW. RETINA; MUSCLE; NERVE.

Being the substance of a communication to the Neurological Society, March 7, 1895.

BY A. D. WALLER, M.D., F.R.S.

THE Weber-Fechner law in its restricted sense designates the relation between magnitude of sensation and magnitude of stimulation. I am using it at present in a more extended sense to designate the relation between cause[1] and effect in living matter, and as instances that I have been able to study I shall bring under your notice experiments upon (1) the retina, (2) muscle, (3) nerve.

The ordinary view of the Weber-Fechner relation between sensation and stimulus presents the former as a logarithmic function of the latter.

Accepting this as in the main correct, and deferring discussion of the departures from the law that take place at the higher and at the lower limits, let us first face this definite question: there is a disproportion between stimulus and sensation such that equal increments of sensation require increasing increments of stimulation, or—what amounts to the same thing—that each equal increment of

[1] The word "cause" is here used in its popular sense, to denote the immediate discharging cause, not in its strict physical sense, when of necessity cause and effect are equal and opposite. Evidently it is only in the first sense of "discharging cause" that the stimulus of a nerve is the cause of muscular contraction, just as in the same sense a spark is called the cause of an explosion, or a match the cause of a fire. The unstable equilibrium of fuel, or of powder, or of nerve-matter is obviously a factor in the aggregate causation, particular elements of which are treated as terminals of thought when, *e.g.*, we consider the relation between magnitude of stimulus and magnitude of contraction, or between magnitude of stimulus and magnitude of sensation.

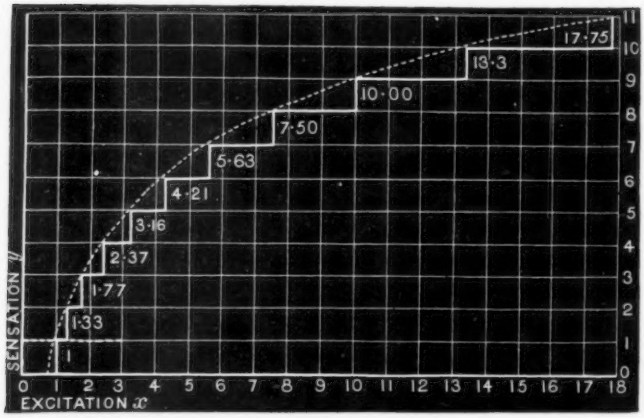

FIG. 1 A.

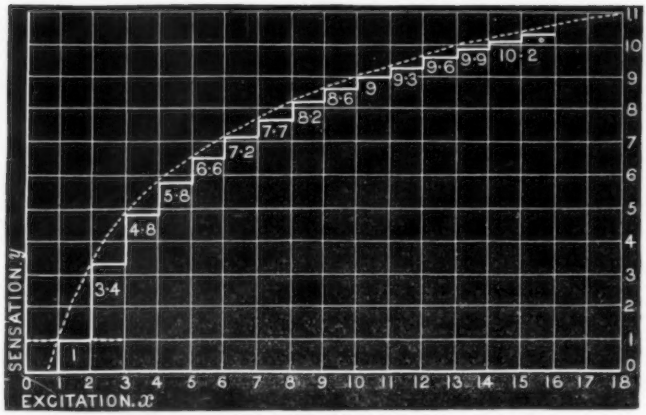

FIG. 1 B.

The Weber-Fechner curve, expressive of a logarithmic relation between excitation (increasing along the abscissa) and sensation (increasing along the ordinates). The two curves, A and B, are identical. In A the curve is obtained by taking along the abscissa the magnitudes of excitation required to produce "the minimum perceptible difference" between successive stimuli assumed by Fechner as the unit of sensation. In B the curve is obtained by calculating the magnitudes of sensation corresponding to an arithmetical increase of excitation. Both curves show, therefore, the same relation, A showing that to equal increments of sensation increasing increments of stimulation are necessary, B that equal increments of stimulation cause diminishing increments of sensation. In A the steps of sensation are equal, but each tread is longer than its predecessor; in B the steps of sensation diminish in the ascending scale, their treads being equal. The curves are constructed from numbers corresponding with an increase of excitation by $\frac{1}{4}$ necessary to produce a perceptible increase of sensation.

stimulation produces a diminishing increment of sensation. Is this disproportion of physiological or of psychological origin?

To present this question clearly it is convenient or even necessary to distinguish two stages in the passage from external physical stimulus to sensation; a not infrequently committed ambiguity in the use of the word "stimulus" will thereby be avoided.

We have:—

(*a*) the external stimulus, *i.e.*, the objective phenomenon or external cause of excitation apart from the excitation itself.

(*b*) the internal stimulus, *i.e.*, the material sensificatory change, provoked in the nervous system by the external stimulus.

(*c*) the sensation itself, *i.e.*, a subjective phenomenon concomitant with the sensificatory change.

Given, *e.g.*,

A series of external stimuli of magnitudes 1, 2, 3, 4, 5, &c., and a series of sensations of magnitudes 1, 3, 5, 6, 7, &c. Does the disproportion take place on physiological ground between external stimulus (*a*) and internal stimulus (*b*), or on psychological ground between internal stimulus (*b*) and sensation (*c*)?

Some contribution towards an answer to this question would be afforded if we could measure and express in any scale of units a series of internal sensificatory changes—effects of a series of external stimuli, their cause.

I.—On these grounds I have bestowed a considerable amount of attention upon the RETINA, where, thanks to the labours of Holmgren, followed by Dewar and MacKendrick, Kühne and Steiner, and others, we know that the stimulus of light produces an electrical change that may be utilised as the objective sign of a physico-chemical change in the retina. It is not proved, but it is highly probable, that such physico-chemical change is the sensificatory phenomenon in so far as the retinal portion of the retino-cerebral organ is concerned. For the purposes of the

present inquiry this may be assumed, and we may proceed to measure out quantity of external stimulus (light), and read off upon the galvanometer quantity of physico-chemical change, considering such physico-chemical change as an instance of internal stimulus or sensificatory phenomenon.

Here is an example of retinal action consequent upon luminous stimuli of increasing magnitude.

The magnitude of stimulation was in this case varied by altering the distance from the retina of a standard candle; the object submitted to experiment was the freshly-excised eyeball of a frog, resting upon one electrode by its posterior surface, which included the cut stump of the optic nerve,

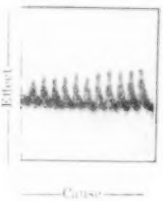

FIG. 2.

Fig. 2.—Galvanometric deflections (*i.e.*, retinal effects) in a frog's eyeball caused by exposures lasting one-eighth of a minute at minute intervals to the light of one candle at the distances 14·1, 10, 7·07, 5·77, 5, 4·47, 4·08, 3.78, 3·53, 3·33, 3·16 feet giving stimuli of the strengths: 0·5, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10 units, the unit taken being equal to $\frac{1}{100}$ of a standard candle at a distance of one foot. The curve obtained by joining the summits of the ordinates closely resembles the Weber-Fechner curve of fig. 1.

led off to the galvanometer by that electrode and by a second electrode, the actual end of which consisted in a saline-moistened thread resting upon the edge of the cornea. The eyeball was adjusted inside a dark box opposite to a blackened tube, through which light was let in or blocked off by the movements of a shutter not connected with the box, moved by hand or by an electro-magnet. The cornea was directed upwards—light impinged upon the sclerotic, glancing over and reflected from the surface of the eye-ball, so that there was no appreciable focussing of the candle flame through the refractive media of the eye. In the

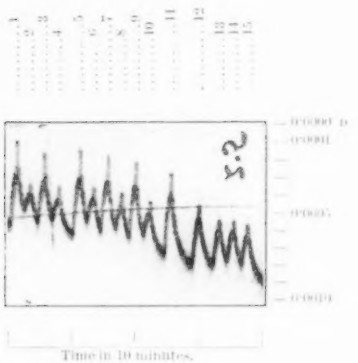

FIG. 3.

Fig. 3.—Galvanometric deflections by retinal currents produced by steady and by interrupted illumination of different intensities.

(1) Steady light at 10 units for 1 min.
(2) Interrupted „ „ „ $\frac{1}{4}$ or 2·5 units.
(3) Steady
(4) Interrupted
(5) Steady
(6) Interrupted
(7) Steady
(8) Interrupted
(9) Steady
(10) Interrupted
(11) Steady at 40 units for 1 min.
(12) Interrupted „ „ $\frac{1}{4}$ or 10 units.
(13) Steady at 2·5 units for 1 min.
(14) „ „
(15) „ „

N.B.—The interruptions of light were caused by a revolving disc with open sectors giving 10 interruptions per sec. The sum of the sectors occupied $\frac{1}{4}$ of the whole disc.

The particular points illustrated in this figure are:

(1) That at equal strengths interrupted light produces a smaller effect than continuous light.

(2) That at unequal strengths the effect of interrupted light at 10 units is about equal to that of steady light at 2·5 units, and of steady light at 10 units to that of interrupted light at 40 units, the open sectors being $\frac{1}{4}$ of the disc.

		Interr. 10.		Steady 2·5.
Compare deflections	2, 4, 6, 8, 10,	with	13, 14, 15	
		Steady 10.		Interr. 40.
„	„	1, 3, 5, 7, 9	with	12

particular experiment of which fig. 2 gives the results, the light was blocked off for seven-eighths of a minute and let on for one-eighth of a minute, during each successive minute, by means of an electro-magnet in the circuit of a battery and a mercury pool at which contact was made by a metal point fixed to a clock wheel and revolving once a minute. These instrumental details need not be minutely described; the apparatus may be inspected in the dark room; its plan is diagrammatically presented in fig. 13.

A simple inspection of fig. 2 shows us that the heights of the ordinates (*i.e.*, the magnitudes of effects) corresponding to an arithmetical increase of causative stimuli along the abscissa, do not form a straight line, but a curve, concave to the abscissa—a curve increasing by diminishing increments. It is similar in character to the classical Weber-Fechner curve given above.

I have also graduated the light by means of a revolving disc, with open sectors, the size of which could be varied. Fig. 3 exhibits the results of such an experiment.

This of itself is, I think, justification enough for an answer to the question formulated a few minutes ago—which I formulated in other terms two years ago—"Is the material sensificatory brain-change proportional to the internal stimulus, and the sensation some geometrical function of that change, or is the material change itself a geometrical function of the stimulus, and attended with sensation in direct proportion to its own magnitude?"[1] I then felt entitled to no opinion of my own, but quoted authorities *pro* and authorities *con* without adding note or comment. But looking to this result, considering that in fig. 2 we have external stimulus along the abscissa, internal stimulus along the ordinate, our judgment must be strongly inclined towards the second alternative.

II.—Let me at once turn your attention to a second item in the case, viz., to MUSCLE.

[1] Introduction to "Human Physiology," 2nd edition, p. 550.

Considering muscle, not for its own special sake, but in a most general light as excitable living matter, of which the chemico-physical response to external stimuli is measurable, we may study as an instance of the relation between causative stimulus and excited effect, the relation in muscle between magnitude of stimulation and magnitude of contraction.

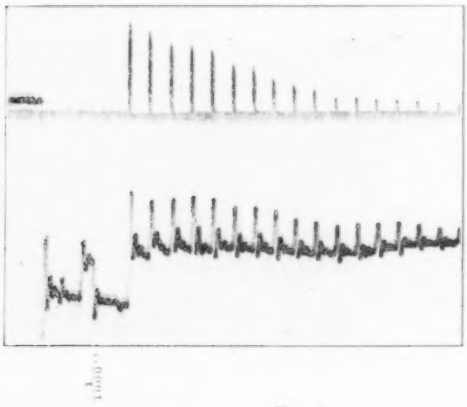

FIG. 4.

Simultaneous tracing of muscular contraction, and of the accompanying current of action. Galvanometer shunt $\frac{1}{10}$. Preliminary upward deflection by $\frac{1}{1000}$ volt.

We might do this galvanometrically, but the galvanometric sign of action in the case of muscle is simply a *replica*[1] of its mechanical response, and the latter is far more simply examined. I have urged elsewhere that as regards mechanical response, it is better to adopt an approximately isometric than an approximately isotonic method.[2]

[1] This is not, strictly speaking, correct; the electrical signs of muscular action are a far more delicate, and in some respects more accurate, quantitative indication than are the mechanical signs; moreover, the electrical response outlasts the *visible* mechanical response.

[2] "The Sense of Effort," BRAIN, 1891, p. 186.

An inspection of the two figures 5 and 6 shows that the general law, diminishing increments of effect (contraction), produced by equal increments of cause (stimulation), is similar to that already observed in the case of sensation, and in that of sensificatory change.

I do not now wish to dwell upon details, nor technical matters, nor historical matters, but before passing on to our next case I should like just to mention that the position here taken up has been considered and studied by other observers. Fick, Hermann, Preyer, Santesson, Cybulski and Zanietowski have handled the question, and Preyer in particular

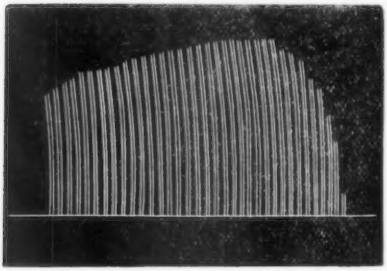

FIG. 5.
(To be read from right to left.)

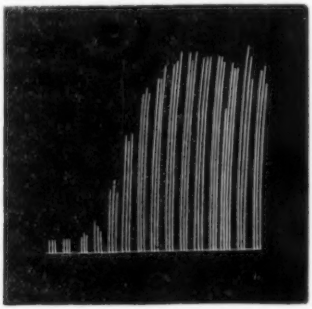

FIG. 6.
(To be read from left to right.)

Contractions of a frog's gastrocnemius in fig. 6, caused by induction shocks of arithmetically increasing strength in fig. 5, caused by the discharges of arithmetically increasing quantity from a condenser.

Fig. 5 exhibits a curve of increasing effect with diminishing increments, viz., at once concave to the abscissa.

Fig. 6 exhibits an S-shaped curve of increasing effects, viz., at first convex then concave towards the abscissa.

has considered it from the general point of view here taken up as indicated by the title of his monograph, "Das myophysisches Gesetz," Jena, 1894, the chief conclusion of which is to the effect that the shortening of muscle varies as the logarithm of the electrical excitation.[1] The other point

[1] And Dubois, in his admirable monograph on *Pholas Dactylus*, urges the same idea on still more cogent data, involving as they do both motion and sensation. In his account the magnitude of the "mouvement avertisseur" giving rise to the dermic phosphene, is a logarithmic function of the magnitude of stimulation, and the magnitude of the reflex retraction of the syphon symptomatic of sensation is proportional to the magnitude of the "mouvement avertisseur," which is the antecedent link in the sensificatory process.—"La Pholade Dactyle," Paris, 1892.

to which I wish to call your attention in passing is one that from a totally different standpoint has been insisted on by Prof. Weldon, and is illustrated in fig. 6.

The ordinates do not give from the outset a diminishing increment leading to a curve concave to the abscissa, but at the very outset increasing increments, and later only the usual diminishing increments. The curve formed by joining the summits of the series of contraction ordinates is S-shaped, beginning convex to the abscissa, continuing concave to the abscissa. And, as we shall see in a moment, a similar mode of commencement is apparent in the case of nerve (*e.g.*, in fig. 7).

III.—The third case to be considered is that of NERVE. Here (if I am not grossly deceiving myself, and have not fallen into some unsuspected form of instrumental fallacy) the results are of the highest interest. On nerve the curve expressing the relation between cause and effect is a straight line, the effect is proportional to the cause, at least within moderate ranges of excitation, exceeding, however, the upper limit of maximum functional effect, as gauged by muscular contraction. I hasten to add a short description of the conditions of experiment. The usual nerve-muscle preparation is laid across two pairs of electrodes; the exciting electrodes from an induction-coil are against the nerve midway between the leading-off electrodes to the galvanometer and the muscle, which in this case is preserved as an accessory indicator of function in the nerve. The electrical effects on the galvanometer and the mechanical effects on the muscle are simultaneously recorded on the photographic plate in a way that would be tedious to describe. It will be obvious if you will glance at the apparatus; its plan is diagrammatically presented in fig. 14. All precautions known to me for the avoidance of the two chief fallacies in such an experiment, viz., electrotonic currents and unipolar action, have been observed.

Fig. 7 exhibits the point I have just stated; the electrical effects on nerve consequent upon stimuli of magnitudes 1, 2, 3, 4, are in the proportion, 1, 2, 3, 4, a point that is still

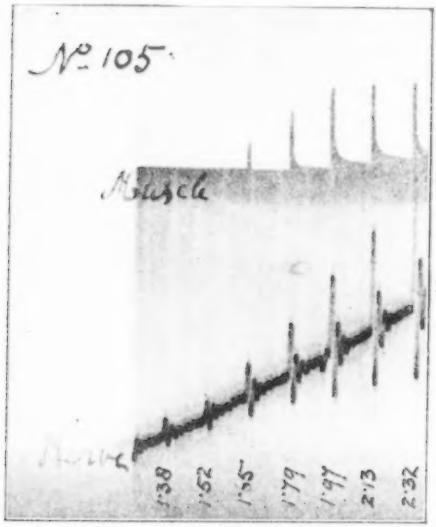

FIG. 7.

	Strength of Stim.	Nerve Neg. Var.	Muscle Lift.
No. 1	1·52 units ..	2·5 mm. ..	0
,, 2	1·65 ,, ..	5 ,, ..	4
,, 3	1·79 ,, ..	7·5 ,, ..	9
,, 4	1·97 ,, ..	12 ,, ..	12
,, 5	2·13 ,, ..	15 ,, ..	12·5
,, 6	2·32 ,, ..	19 ,, ..	12·5

Simultaneous tracing of the galvanometric and muscular effects of nerve excitation. The nerve of a nerve-muscle preparation led off to the galvanometer by unpolar electrodes to its central end, is excited by tetanising currents at intervals of 1 min. for periods of $\frac{1}{2}$ min. No galvanometer shunt; each centimetre of swing = about $\frac{1}{10000}$ volt.

N.B.—The current of injury of the nerve was such as to deflect the spot downwards as regards this tracing; the upward inclination of the galvanogram indicates the usual decline of that current; the upward black lines across the galvanogram are due to the negative variations, the subsequent downward black lines are due to after-oscillations of the magnet and mirror. The plan of experiment is shown by fig. 14, but as printed the tracing is reversed, *i.e.*, the muscle shadow was south of the nerve deflection, and the current of injury was north (cut end of nerve to north terminal) and the negative variation south.

more evident in the diagram fig. 8, constructed from corrected data in continuation of the experiment, part of which is given in fig. 7.

The chief positive results have been laid before you, and will have to be matured by further experimental criticism. The main question of the Weber-Fechner relation between stimulation and sensation is touched by them at more than one of its facets. The more general question of the

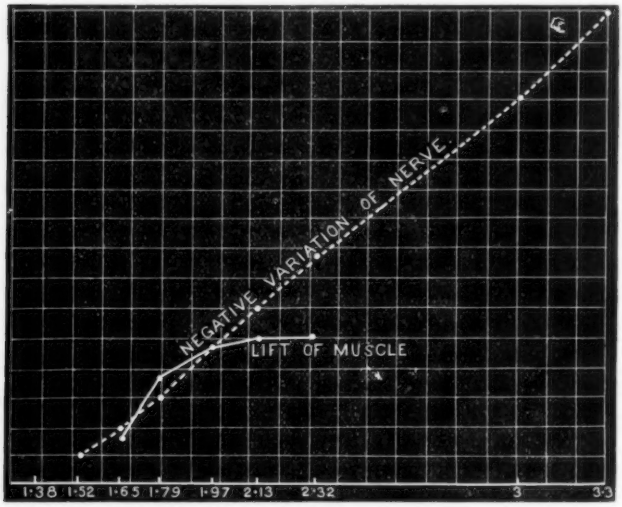

FIG. 8.

Stimulation units along the abscissa; electrical effects in nerve, and simultaneously observed mechanical effects of its muscle, along the ordinates.

relation between physical cause and physiological effect has *ipso facto* been involved. And as in any consideration of phenomena *de tactu*, side-views of phenomena accessory to the main inquiry have presented themselves, and glimpses of imperfectly viewed relations—what may be termed subpositive considerations—have also offered themselves to mind. In this connection the following points seem to me to be the more considerable :—

(1) The S-shaped curve of muscle exhibited, *e.g.*, in fig. 6—is it peculiar to muscle, or to muscle as representative of living matter? Will it find itself repeated in a Weber-Fechner sensation curve? I am unable to say. I have no *data* of my own with regard to that most difficult investigation, viz., that of the sensation curve low down, as it is rising from the threshold of stimulation. But I would ask you to consider probabilities. Which should we prefer—to have maximum increments of sensation to equal increments of stimulation low down in the scale close to the threshold, or higher up in the scale within the working range of the stimulation experienced in ordinary life? The first alternative would, it seems to me, be characteristic of an almost intolerable state of hyperæsthesia in relation to the tumult of minute stimuli by which we are surrounded. I should prefer to be endowed with an S-shaped curve. And this is not merely a personal preference; there are one or two significant hints from various sides pointing towards the conclusion that an S-shaped curve is typical of the response of living matter, whether by quantity of sensation, or by quantity of motion, or by quantity of loss of motion, in response to external stimulation, to physiological accidents and to pathological injury.

It is only in the medium range of the scale of sensation that the logarithmic curve comes out. Hering and his pupils, working at lower ranges, found that the curve was approximately a straight line. Considering the enormous difficulties attendant upon such experiments, it is easy to believe that the initial tail of an S should present itself to the observer as a straight line. In the case of a muscle—if it is allowable to regard muscle as a representative of living matter, and the physico-chemical changes taking place in it in consequence of stimulation as analogous with the sensificatory physico-chemical changes taking place in the living matter of sensory organs—we have this S-shaped curve outward and visible. And on this same living matter—muscle—the decline at death and the decline in fatigue, as far as I have been able to judge, run this S-shaped

course, only the S of the waning is as the S of the waxing reversed—as if viewed in a mirror.[1]

(2) The second most thought-arousing point is with regard to the electrical signs of nerve activity—of the nerve-fibre, not of the nerve-cell. I pointed out some ten years ago, quite independently of the observations of Wedenski and Bowditch, that nerve-fibre is practically inexhaustible, a force-transmitting, not a force-transforming organ, a passive physical conductor. I then made certain reservations as regards the experimental fallacy of electrotonic currents, which have been removed by further investigation, and the conclusion is at present an admitted one. Well, the conclusion being admitted, I do not think that it need surprise us to find in such an organ the physico-chemical effect simply proportional to the physico-chemical cause.

I hasten to add that it is only within a certain range—a range far exceeding, however, the physiological working-range of nerve—that the simple proportion holds good. Many more experiments directed to this particular point will be required before I shall be able to lay an entire curve before you with any justifiable degree of confidence. All I am entitled to say at present is that within what may be termed physiological limits, the curve expressing the ratio between cause and effect is a straight line; the sub-minimal and supra-maximal ends of that curve require further experimental determination; as far as my experiments have yet gone it has appeared to me that the beginning of the curve is convex to the abscissa, the end of the curve concave to the abscissa.

[1] Without unduly insisting upon its possible significance, the fact is perhaps worth noting that an ordinary simple contraction of muscle due to a single stimulus, consists in each of its two parts of an S-shaped curve; in the period of ascending energy, the rise is by increments increasing, then diminishing, in the period of descending energy the fall is by decrements at first increasing then diminishing, giving ∫∿. The rising and falling effects have respectively their immediate cause in the muscle; it may be that each such cause is of uniform speed, in which case the effect is slow—quick—slow with uniform cause; or it may be that each such cause is itself slow—quick—slow, with its effect correspondingly slow—quick—slow; but even in this case there is an S-shaped relation between cause and effect, inasmuch as the immediate cause is itself an effect of a previous cause, the instantaneous stimulus provocative of decomposition of matter and evolution of energy, during the rise followed by what may be regarded as a negative state, provocative of recomposition and involution of energy during the fall.

Considering only that part of the nerve curve corresponding with the functional range between minimal and maximal muscular effects, we have a straight line, below which a short initial tail is convex to the abscissa, and above which, after a further prolongation of the straight line, the curve tends to become concave to the abscissa. An entire curve may then be considered as composed of three portions: (1) a short subminimal portion, convex to the abscissa, increasing by increasing increments; (2) a long straight middle portion, inclusive of the functional range between minimal and maximal muscular effect, increasing by equal increments; (3) an ultra-maximal portion far above the values of maximal functional effects, concave to the abscissa, increasing by diminishing increments.

It is not possible to exhibit these several features in any one line drawn to scale. To bring out the first portion of the curve the scale must be chosen very large, when of course the second and third portions are lost to view; to bring out the third portion the scale must be chosen very small, when of course the first portion is altogether indistinguishable. On an intermediate scale, the functional portion of the second portion is best shown, *e.g.*, in fig. 8. The first portion is visible in fig. 7. The third portion can be constructed from the numbers of fig. 9.

(3) A third point is subsidiary to the above. It will be most clearly exposed to you by the examination of an actual experiment (*e.g.*, fig. 9), viz., that far beyond that point where stimulation of the nerve gives a maximal effect upon the muscle, the effect upon the galvanometer of the electrical change in the nerve continues to increase. Due care was observed to exclude the unipolar and the electrotonic fallacies; the electrical effects were unchanged in direction with reversed direction of the excitation, viz., they were true "negative variations," the electrical concomitants of excitatory changes in the nerve. This being so, we are bound to recognise that the magnitude of an excitatory effect in nerve-conductors provoked by electrical excitation, is no index to the magnitude of functional disturbance within what may be regarded as physiological limits.

Method.—The most essential feature of the method employed in the above observations was the use of photo-galvanographic records, by means of an apparatus (galvano-

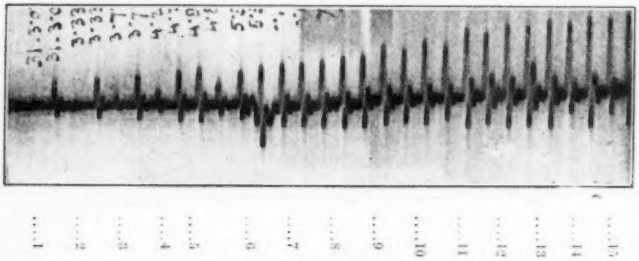

FIG. 9.

Continuation of Obs., 105. Galvanometer shunted $\frac{1}{10}$.

	Stimulation strength.		Galvanometric deflection.
No. 1	3·02 units	....	35 mm.
„ 2	3·33 „	....	40 „
„ 3	3·72 „		
„ 4	4·25 „	....	50 „
„ 5	4·80 „		
„ 6	5·50 „	....	60 „
„ 7	6·10 „		
„ 8	7·00 „	....	70 „
„ 9	8·20 „		
„ 10	9·60 „	....	85 „
„ 11	19·20 „		
„ 12	28·75 „	....	105 „
„ 13	38·30 „	....	110 „
„ 14	47·90 „	....	110 „
„ 15	57·50 „	....	110 „

N.B.—The values given for these galvanometric deflections are of necessity inexact, inasmuch as measurements of ordinates, the magnitudes of which have been tenthed by the shunt, and their subsequent multiplication by ten must give rise to a considerable error. But this error does not disguise the main fact apparent on simple inspection of the fig., viz., that the negative variation of nerve continues to increase with increasing strength of stimulation long beyond that strength which has produced a maximal muscular effect as shown by the preceding figure. The deflections to which numbers are not attached were caused by stimulation with reversed direction of current at the respective strengths indicated by the numbers, to exclude the suspicion of electrotonic currents.

graph) already described some years ago in another place,[1] modified in the present instance by the substitution of a sensitive plate for the sensitive paper previously used. The

[1] *British Medical Journal*, July, 1885, p. 135.

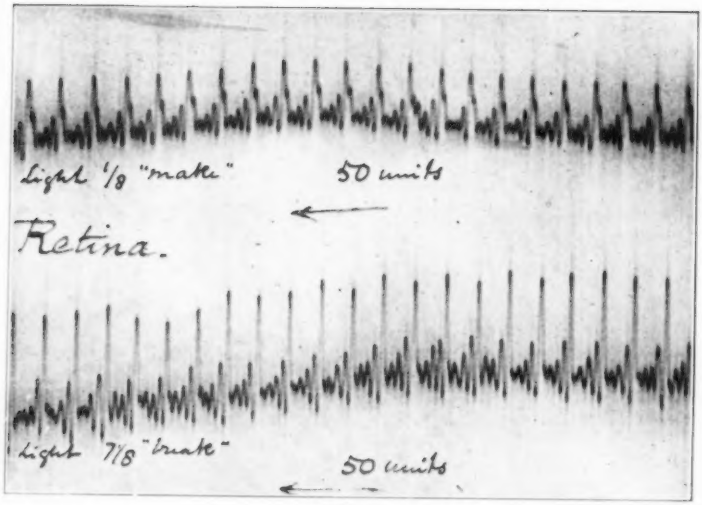

FIG. 10.

Currents of action from frog's retina six hours after excision of eyeball. The upper line is a series obtained by illumination for $\frac{1}{2}$ minute repeated each minute. Owing to the oscillation period of the magnet and mirror the deflection at "make" (about 4 mm.) is interrupted and augmented by the subsequent deflection at "break," the point of "break" is signified by the black spot of the large rise; the oscillations after the large rise are due to the swings of the magnet as it comes to rest. No galvanometer shunt. Each centimetre swing = about $\frac{1}{10000}$ volt. The lower line is a series obtained by illumination for $\frac{7}{8}$ minute repeated each minute. Only the "break" effects are visible, the "make" effects are not separately distinguishable. Oscillations of the magnet as before.

These series illustrate the facts that (1) the effects at "break" of light are greater than those at "make" of light; (2) the effects at "make" and at "break" do not sensibly diminish with repetition.

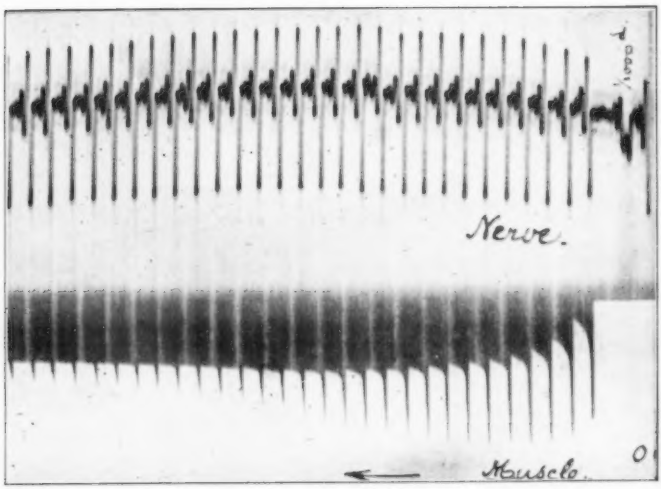

FIG. 11.

Simultaneous record of currents of action in nerve (upper line), and contraction of muscle (lower line), in accordance with the plan of experiment outlined in fig. 14. Tetanisation for $\frac{1}{2}$ min. repeated every minute. Galvanometer shunt $\frac{1}{5}$; preliminary downward or south deflection by $\frac{1}{1000}$ volt. Cut end of nerve to north terminal of galvanometer. Strength of coil 10 units. The electrical effects of nerve action do not sensibly diminish while the mechanical effects of the supplied muscle suffer the usual fatigue decline.

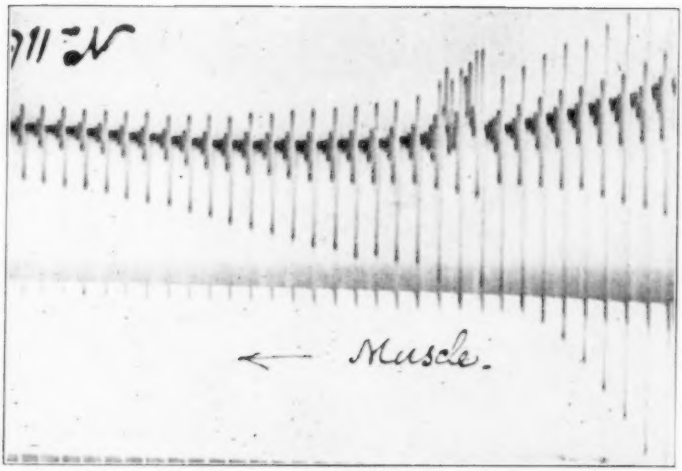

FIG. 12.

Simultaneous record of currents of action in muscle (large swings downwards, viz., south, the tendon being connected with the north terminal of the galvanometer, followed by the usual oscillations of the magnet as it comes to rest) and of the contractions of the same muscle. Galvanometer shunt $\frac{1}{4}$. Each centimeter swing = about $\frac{1}{1000}$ volt. Strength of coil 20 units. Tetanisation for $\frac{1}{8}$ min. repeated each minute.

N.B.—The muscular contractions (downward black teeth from the black border) are at first overrun and rendered indistinct by the large deflections of the magnet. The irregularity in the series was accidentally caused by the fouling of a wire.

The electrical effects of muscular contraction decline with the decline of the mechanical effects. The former are a more delicate index of the state of muscle than the latter.

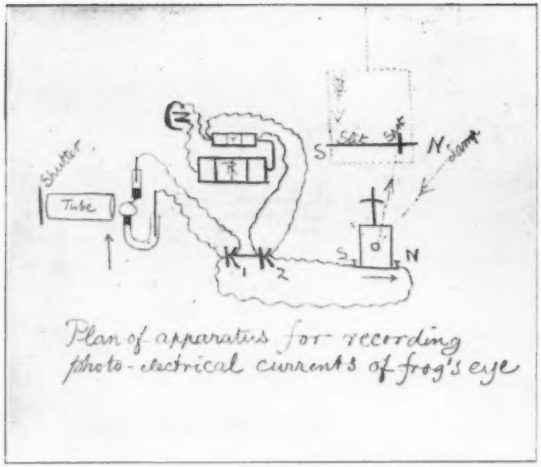
Shutter
Tube
K1
K2
S
N
Slit
Lamp
Plan of apparatus for recording photo-electrical currents of frog's eye

Fig. 13.

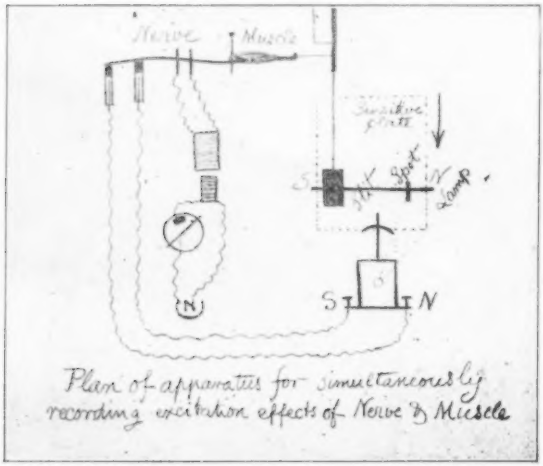
Nerve
Muscle
Sensitive plate
Slit
Lamp
S
N
Plan of apparatus for simultaneously recording excitation effects of Nerve & Muscle

Fig 14.

entire complexion of enquiries of the kind is altered when the photographic method is adopted; points that escape the closest scrutiny in its absence, come into striking evidence, and doubtful readings are replaced by peremptory writings—autographs of the objects under observation.

Seeing that the intensity of light falling upon the eyeball varies inversely as the square of its distance, I prepared a scale graduated in units, in accordance with the following scale of numbers, choosing for the unit the $\frac{1}{100}$ part of a standard candle at a distance of 1 foot, or 1 candle at a distance of 10 feet.

Distance.	Strength.
1 foot	100 units.
1·15 „	75 „
1·41 „	50 „
2 „	25 „
2·29 „	20 „
2·58 „	15 „
3·16 „	10 „
3·33 „	9 „
3·53 „	8 „
3·78 „	7 „
4·08 „	6 „
4·47 „	5 „
5 „	4 „
5·77 „	3 „
7·07 „	2 „
10 „	1 „
11·5 „	0·75 „
14·1 „	0·50 „
20 „	0·25 „

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ON THE NATURE OF THE PHYSIOLOGICAL ELEMENT IN EMOTION.

BY PROF. A. E. WRIGHT, M.D.

Army Medical School, Netley.

It is the signal merit of Prof. James to have recognised that the physiological element is the predominant element in the emotions, and that the problem of the nature of the emotions is essentially a physiological problem. The following study of the emotions consists in an attempt to apply to the solution of that problem the more adequate conceptions of the spinal centres and the somatic and visceral nerves which we owe to the researches of Gaskell.

Obviously the easiest way to approach the study of the emotions is to proceed by the method of physiological experiment.

An appropriate experiment would consist in taking a kitten and in noting the changes that occur in it when it is suddenly confronted with a strange dog. The emotional stimulus which would thus be applied would evoke a whole series of reflexes. The rhythm of the kitten's respiration would be altered, its heart-beat would be affected, the size of its pupils would be altered, and the hairs upon its tail would stand on end. All these would be reflexes upon visceral efferent nerves. Superadded to these involuntary visceral reflexes there would be reflex responses upon the semi-involuntary facial muscles; the lips would probably be drawn back so as to show the teeth. And, lastly, there

would be reflex responses in the voluntary muscular system; the kitten would assume an attitude of self-defence.

The essential features of this emotional reaction would appear to be (*a*) the origination of the emotion in a violent sensory stimulus; (*b*) a resulting condition of extreme neural tension in the reflex centre; and (*c*) an outflow of neural energy, primarily, into the efferent paths associated with involuntary muscles, secondly, into the efferent paths associated with semi-involuntary muscles, and thirdly, into the efferent paths associated with voluntary muscle.

What holds true of the emotion which is evoked in the kitten by the encounter with the dog holds true of all other emotion. Wherever we have a condition of high neural tension, followed by reflexes upon involuntary muscle, we are dealing with emotion.

The descriptions which the poets give of emotion are in evident agreement with our definition. Two will suffice. We have Dante's "io ch'avea d'orror la testa cinta," and Juliet's "hood my unmanned [*i.e.*, untamed] blood bating [*i.e.*, beating as a hawk's wings beat] in my cheeks." Each of these descriptions of emotion tells of a condition of high neural tension, and of an outflow of neural currents into the involuntary muscular system. The "girdling of the head with horror" would indubitably be a function of the overflow of neural energy into the cervical sympathetic nerves, for Sherrington has demonstrated that the stimulation of the cervical sympathetic nerves[1] in the macaque produces the erection of a fringe of hair upon the head, and gives the monkey a terror-struck appearance. Similarly in the case of Juliet, the condition of emotion is associated with high neural tension and visceral reflexes. The latter result in tumultuous heart-beats and vaso-dilatation.

Moreover, the results of clinical observation accord well with our physiological inferences, for we find that heart

[1] Langley similarly showed that the erection of hairs on a cat's tail was brought about by the stimulation of the sympathetic fibres. On the other hand, as my friend Prof. Sherrington has pointed out to me, the labial vibrissæ in the cat are innervated, not by sympathetic fibres, but by branches of the fifth. This is exactly what we should expect *a priori*, for the erection of the vibrissæ of course occurs quite independently of any emotional reaction.

disease, Bright's disease, jaundice, and dyspepsia are occasional sequelæ of profound emotional disturbance. The emotional reflexes have in these cases evidently been correlated with serious nutritional disturbances of the viscera.

We must not, however, suppose that these visceral reflexes constitute the physiological essence of the emotion.[1] The physiological essence of the emotion is the high neural tension in the reflex centre, and not the visceral reflexes that result from that condition. For the psychological phenomena of any emotion precede the manifestation of that emotion, and they are coincident, not with the visceral reflexes, but with the antecedent neural tension in the reflex centres.

Having thus obtained a definition of emotion which conforms to the data of our physiological and psychological experience, we may pass on from this to consider the transformation of the reflexes that occurs when the stimuli which originally evoked emotions lose, as all stimuli gradually do, their emotional potency.

This transformation is a question of a replacement of the generalised somatico-visceral reflex by a specialised somatic reflex.

It is a transformation which is in gradual process all the time the child is growing up into the adult. In childhood the reflexes approximate to the primitive generalised type of reflex. There is as yet no differentiation into somatic and visceral reflexes, and every afferent phenomenon evokes a response both upon voluntary and upon involuntary muscle. In all such reflex responses there is therefore an element of emotion. When, however, the child grows older, the outflow of neural energy which occurs in response to the ordinary afferent impulses becomes more and more restricted to the somatic efferent channels. And gradually, hand in hand with this elaboration of the purposive voluntary action,

[1] We may perhaps note that the child does not discriminate between the essence of the emotion and its manifestation. The unsophisticated child will, for instance, say "Now you have made me *cry*." He will never say "Now you have made me *sorry*," for he does not attend to his psychological condition. He attends only to the results of his reflexes.

the emotional visceral element[1] fades out from the reflex process.

An example or two will serve to show what is involved in this substitution of a somatic for the primitive somatico-visceral reflex. When a tyro-sportsman goes out for the first time after game, the sight of a rabbit jumping up before him evokes a considerable volume of emotion. This expresses itself in a violent start, and in tumultuous heart-beats. When, however, the sportsman has become familiar with these sensations, and when he has practised the voluntary responses which are appropriate to such an occasion, the jumping up of a rabbit will no longer evoke an emotional reaction. It will evoke, instead, the purposive reflex responses which are associated with aiming and firing.

Similarly a surgical operation will evoke a considerable emotional reaction in any unaccustomed onlooker. But when a medical student sets himself to learn his surgical work, he learns to respond to such sensations with the appropriate somatic reflexes. And when the adaptation of these purposive voluntary responses to the sensory phenomena of the operating table has been achieved, the student, who now merits the title of "*surgeon*," finds that the nerve currents "no longer keep running down into his viscera."

We must, however, note that the education of the reflexes is never even tolerably complete. When any startlingly novel phenomenon—any sensation in short to which a voluntary reflex movement cannot be immediately adapted—presents itself, the neural tension will overflow with full force into the involuntary muscular system, and particularly into the respiratory system. We therefore

[1] The fading out of this emotional element from the reflex process is a frequent theme in Wordsworth's poetry.

It is, for instance, the theme of his "Intimations of Immortality from Recollections of Early Childhood." The "something that is gone" is the visceral reflex. The bringing down of all life to the lower level of the somatic reflex is the "closing of the prison house upon the growing boy." Childhood is "the hour of splendour in the grass and glory in the flower." Manhood is the time when the "celestial light" which originally illuminated every common thing has completely faded out "into the light of common day."

speak of such a startling sensation as something "that takes the breath away."

What has been effected, either by natural processes or by education is, therefore, not a suppression of the tendency of the neural tension to overflow into the visceral nervous system, but rather a diversion of the nerve outflow into the somatic efferent channels in the case of a certain number of constantly recurring sensations. And we must note that this more effective outflow through the somatic efferent channels goes hand in hand with a diminution of the neural tension in the reflex centres.

Very obvious physiological advantages accrue from this transformation of the reflex processes. *First*, the neural energy will no longer run to waste in visceral reflex movements. *Secondly*, when the visceral reflex is eliminated there will no longer be any danger of the visceral commotion, which is initiated by that reflex, reacting in an unfavourable manner upon the reflex centres. *Lastly*, when the high neural tension is abolished in the centre, the conditions will obviously be more favourable to the delicate adjustment of the reflex responses upon voluntary muscle. The advantages from the point of view of practical utility need not be further insisted on.

Considerations of utility do not, however, effect physiological changes. We have, therefore, still to seek for the effective physiological motive force which brings about this transformation of the reflexes. As a preparation for undertaking this task, we must, however, consider, *first*, the *a priori* necessity for some systematic control of the reflexes, and, *secondly*, the nature of the provision which actually exists in the organism for the exercise of that control.

The *a priori* necessity for some system of control becomes obvious when we consider that if each minimal stimulus were to evoke a separate reflex movement in an organism which was endowed with a sensitiveness at all approaching that of the human organism, life would be a mere chaos of muscular movement. If there were no storing away of nerve-energy there would be no continuity of purpose, and no possibility of making the present subserve the requirements of the future.

It is, however, precisely this continuity of purpose, this making provision for the future, this "looking before and after," which is the specially characteristic note of human life. And of all this an effective control of the reflex processes is the precondition.

In the case of the reflexes upon voluntary muscles, the control, which we have seen to be so essential, is exercised almost exclusively through the medium of the inhibitions. These are of course continuous in their operation. And so effective is this control that a stimulus cannot, even in the most sensitive organism, evoke a movement of voluntary muscle, unless it can first effect the lifting off a considerable dead weight of inhibition from the reflex centres.

In the case of the visceral reflexes inhibitory control plays a comparatively subordinate rôle. But on the other hand, the reflex centres are so constituted that no outflow of neural energy can take place into visceral efferent nerves until a very considerable neural tension has been achieved in the reflex centre. This provision operates as a very effective check upon the visceral reflexes.

The voluntary and the visceral reflexes are thus sharply contrasted. The voluntary reflex movements (*a*) correspond to low tension discharges from the centre (*b*) the mechanism which controls them is extraneous to the spinal reflex centre, and (*c*) the control of that mechanism can be relaxed. The visceral reflexes, on the other hand, correspond to high tension discharges from the reflex centre, and the mechanism that controls them is to a large extent intrinsic in the centre.

We have confirmation of this in the fact that a very different intensity of stimulus is required to evoke, on the one hand, a reflex on voluntary muscle, and on the other hand, a visceral reflex. We react with a movement of voluntary muscle to even the lightest touch, but when we react with a visceral reflex, it is always either a question of a very intense form of stimulus, or of a long series of stimuli which have gradually summated themselves in the centre. We may, for instance, contrast the facile culmination of our ordinary uninhibited somatic reflexes with the prolonged

molimen which precedes the culmination of the reflex process in either the vomiting or the ejaculatory centre. It is always easy to initiate any voluntary muscular movement that may occur to us, while it is always difficult to call up an emotion *ex æquo*.

Again, we have clear indications of the high neural tension which is associated with the more important visceral reflexes in the fact that the summation of stimuli which leads up to the reflex action is attended with an outflow of energy along vasomotor and other visceral nerve channels. This is no doubt to be interpreted as a leaking[1] out of the nerve energy which is being accumulated at high tension in the reflex centre. We may take as an example the flushes and shivers and salivation which manifest themselves when the neural tension is rising in the vomiting centre.[2]

Lastly, the enormously greater neural tension, which is associated with visceral as contrasted with somatic reflexes, can be inferred from the intensity of the physiological urgency which precedes the culmination of a visceral reflex.

We here touch the central point of our problem. All high neural tension is attended with a sense of distress.[3] It is this physiological urgency, and not any calculation of utility which presides over the transformation of the condition of high neural tension which is associated with

[1] This leakiness of the reflex centres must obviously operate as an effective drag upon the visceral reflexes. For wherever a certain interval of time is allowed to elapse between stimulus and stimulus the neural tension of the centre sinks to zero again, and the whole process of summation has to be taken under practically *de novo*. This is a fact which is in the experience of nearly everyone, not only after a rough cross channel passage, but also in connection with emotional disturbances in other centres.

[2] The distinguishing mark of what are spoken of in physiological text-books as "centres" (*e.g.*, the centre for vomiting, the centre for micturition, the centre for respiration) is the fact that these are high tension (visceral), as distinguished from low tension (somatic), reflex centres.

[3] It is this sense of general neural distress, and not actual pain, that is the true physiological opposite of pleasure. All neural distress is associated with a condition of high neural tension, and all such neural distress urges forward to the euphoria, which is associated with the condition of neural equilibrium. It is the rebound from the condition of neural distress to the condition of neural equilibrium which constitutes, as indeed Schopenhauer discerned, the essence of pleasure.

emotion to the condition of low neural tension which is associated with purposive action. It is, for instance, not a consideration of utility which urges on a nervous woman to try to learn to cross over a crowded thoroughfare without an emotional reaction. But it is the feeling of distress which is associated with the condition of high neural tension which she seeks to evade. It is with a view to compassing this object that she strives to respond with unhesitating purposive voluntary actions to the multiform stimuli of the street crossing.

We have thus been able to discern what the compelling force is that brings about a gradual supersession of the generalized somatico-visceral reflexes of inexperience and childhood by the specialized purposive reflexes of experience and adult life. We have seen that it is not so much a question of substituting one variety of reflex for another, as it is a question of substituting a condition of low neural tension for a condition of high neural tension.

Now we cannot conceive any other means by which this transformation could be effected except by a more prompt and more adequate outflow of energy through the low tension somatic efferent channels. And such freer outflow of neural energy is almost certainly obtained by facilitating the somatic reflex responses of the spinal centre by a prompt relaxation of the inhibitions which are continuously passing down from the higher to the subordinate centres.

All these points in the physiology of the emotions can readily be illustrated if we allow ourselves to compare the complex of reflex centres, which Gaskell has taught us to group together under the concept of a "complete spinal segment," to a water cistern.

In accordance with Gaskell's scheme, such a water cistern would evidently be supplied by two inflow pipes, corresponding to the somatic and visceral inflow, and with two outflow pipes, corresponding to the somatic and visceral outflow.

Now these outlet pipes will be situated at different levels. The low level outlet will correspond to the efferent paths to voluntary muscle, and the high level outlet will correspond

to the efferent paths to the visceral system. We have further to imagine, first, that the low level outlet was primitively partially obstructed by a valve, and secondly, that this valve has subsequently been geared up to a mechanism which can raise and depress it in such a manner as to completely free or to completely obstruct the flow of water through the lower outlet.

The primitive somatic and visceral reflexes of the child will now correspond to (*a*) a rapid inflow of water; (*b*) the development of a considerable head of pressure in the cistern; and (*c*) an outflow through the low level outlet, and, because of the insufficiency of this outlet, also (*d*) an outflow through the high level outlet.

The transformation of the reflex processes in the direction of a freer outflow into the voluntary muscular system will correspond to such more ample outflow through the low level outlet as will result from a removal of the inhibitory obstruction.

If we now make the further assumption that a considerable strain would be put upon the cistern by the pressure of the full volume of water, we shall be able to realise the utility of a provision for increasing the lower level outflow.

Our illustration will also bring it home to us that the undeniable utility of a provision for an ampler low level outflow is something totally distinct from a physical force such as would necessarily effect an alteration in the system of outflow.

Not only will our illustration make this plain, but it will supply us with something which is completely analogous to the physiological force which is ever tending to bring about a substitution of a somatic for a visceral outflow. The analogous force in the case of our cistern is obviously the pressure of the water, and we can easily conceive methods by which this pressure of water could be made to operate as a constant force in the direction of the removal of the valvular obstruction from the lower outlet.

Again, we can represent to ourselves the emotional results of violent, or sudden, or unfamiliar stimuli if we assume that the mechanism that controls the outflow

through the lower outlet has only been adapted to the circumstances of the ordinary inflow, and that it is inadequate to control the rise of water in the cistern under the circumstances of any unusual inflow. Any such defect of adaptation would obviously bring about an outflow of water through the high level outlet.

Lastly, if we suppose our cistern to leak, and to leak through wider openings as the water pressure increases, we shall evidently constitute a further analogy between our water cistern and the spinal nerve centre.

ON THE FLOCCULUS.

BY ALEXANDER BRUCE, M.A., M.D.

THE flocculus becomes differentiated as a lobule, distinct from the rest of the cerebellum, at a very early period of intra-uterine life—almost contemporaneously, according to Michalcovicz, with the appearance of the middle lobe. Its peduncle also becomes comparatively soon myelinated, some fibres being so at the sixth month, and many more during the seventh month. This development occurs at the time when there are no myelinated fibres in the cerebellum, with the exception of the various constituents of the restiform body and the superior cerebellar peduncle. The peduncle of the flocculus, both at this early stage and throughout the rest of intra-uterine life presents such a striking contrast to the various strands in its neighbourhood that it is somewhat remarkable that so little attention has been paid to its connections as appears to have been done.

The terminations of this peduncle have been very variously described or altogether overlooked by recent writers on nervous anatomy.

Thus Edinger makes no reference to it in his "Bau der central Nervös-organen," although Obersteiner ("Central Nervous System," English translation, p. 304) states that he (Edinger) traces it into the chief auditory nucleus of the opposite side.

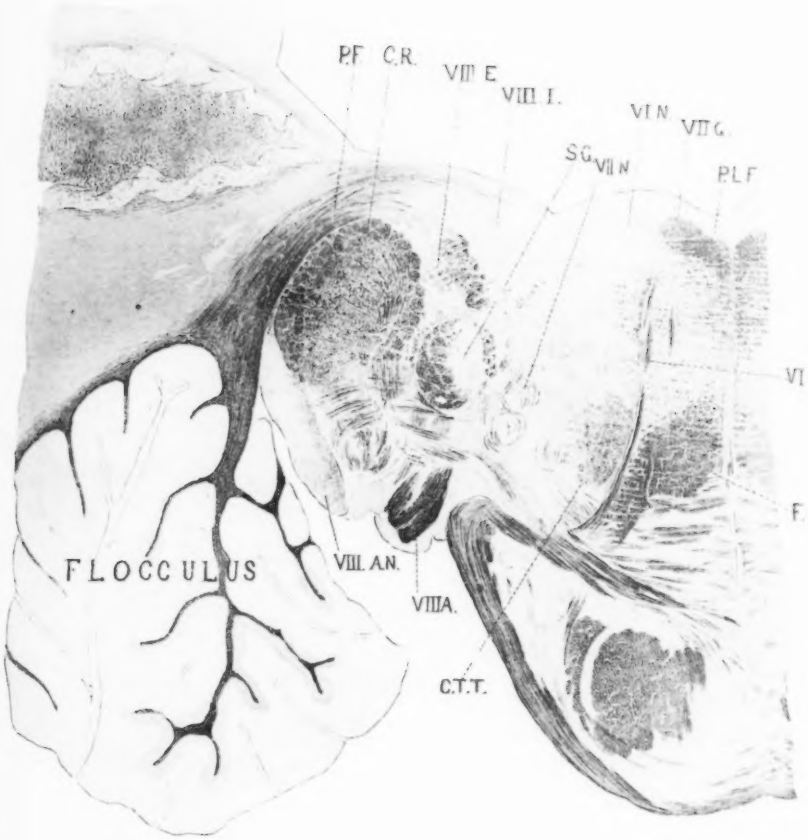
Obersteiner (*loc. cit.*, p. 318) traces the flocculus into the brachium conjunctivum as far as the locus cœruleus. Vegas (*Archiv für Psychiatrie*, vol. xvi., p. 200) traces some of the fibres of the flocculus into the brachium conjunctivum. Bech-

terew, in "Die Leitungsbahnen im Gehirn und Rückenmark" (p. 137), traces its fibres along the roof of the fourth ventricle to the superior vermis. More recently Stscherbak (*Neurologische Centralblatt*, 1893, p. 227) traces part of its fibres to the vermis, along a path which passes between the inner division of the corpus restiforme and the nucleus dentatus; then along the outer side of the nucleus dentatus towards the nucleus globosus and embolus and the cortex of the vermis. Some of the fibres apparently form part of the commissure above the roof nucleus and pass to the opposite flocculus. The second portion passes inwards as far as the upper end of the chief nucleus, where it divides into fine fibres and disappears.

Stscherbak's paper is unfortunately not accompanied by any illustration, and his description is consequently somewhat difficult to follow, but it appears to me that the strand described as passing backwards to the vermis is probably not connected with the flocculus at all, but is really the bundle which passes from Deiter's nucleus along the lateral wall of the fourth ventricle towards the roof nuclei.

In 1892, in my "Illustrations of the Mid and Hind Brain," I stated that the flocculus passes inwards towards the lateral angle of the fourth ventricle and that the majority of its fibres pass inwards and enter into connection with the external and internal nuclei of the auditory nerve and also with the nucleus of the sixth nerve of the same side. The accessory nucleus is also connected with it by numerous fine fibres. It is probable that some fibres of the peduncle also enter into the nucleus dentatus and that another strand passes in an ascending direction along the side of the fourth ventricle. In fig. 7 the connection of the peduncle with the sixth nucleus is indicated. To have made this diagram complete, connections with both the auditory nuclei should have been shown. Indications of the position of the peduncle are shown in plates 9 and 10.

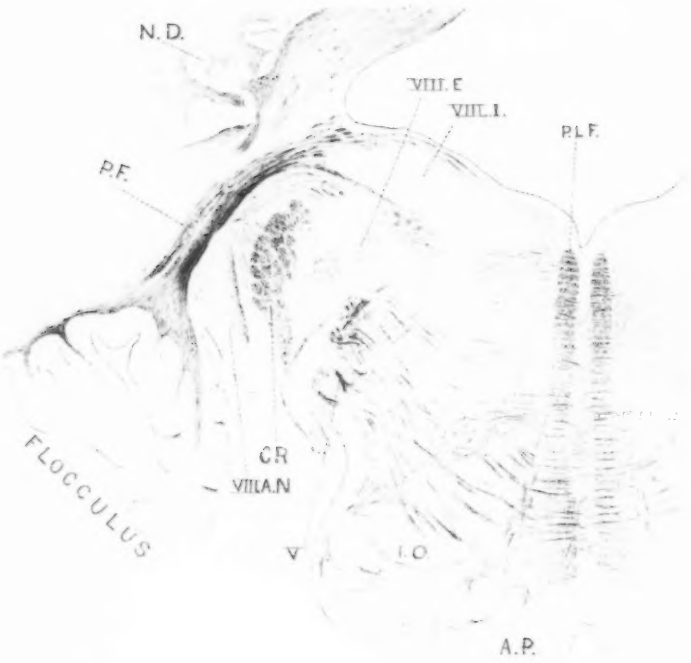
One reason for the variety of the opinions expressed is, I think, the manner in which the flocculus peduncle makes its entry into the side of the pons. If one makes a series of perfectly transverse antero-posterior sections of this region,



DESCRIPTION OF FIGURE 2.

Obliquely Transverse Antero-posterior Section of the Lower Part of the Pons made at an angle of 50 to 55° with the plane of the Raphé, the Mesial Part of the Section being at a higher level than the Lateral.

VII. N. = Nucleus of the facial nerve.
VII. G. = Genu of the facial nerve.
VIII. A. = Vestibular root of the auditory nerve.
VI. = Sixth nerve.
C. T. T. = Central tegmental tract.
F. = Fillet.
S. G. = Substantia gelatinosa, with the ascending root of the fifth to its outer side.
The other letters as in fig. 1.



DESCRIPTION OF FIGURE 1.

PERFECTLY TRANSVERSE ANTERO-POSTERIOR SECTION OF THE UPPER PART OF THE MEDULLA AND THE FLOCCULUS.

P.F. = Peduncle of flocculus passing towards the lateral angle of the fourth ventricle; the continuation of fibres underneath its floor and through the external auditory nucleus has been partly filled in from adjacent sections.

VIII. A. = Accessory nucleus of the cochlear root of the auditory nucleus.

VIII. E. = External auditory nucleus.

VIII. I. = Internal " "

C. R. = Corpus restiforme.

V. = Ascending root of the fifth nerve.

N. D. = Nucleus dentatus. To its inner side part of the superior cerebellar peduncle.

P. L. F. = Posterior longitudinal fasciculus.

A. P. = Anterior pyramid.

I. O. = Inferior olive.

Note.—The level of the posterior part of the section is slightly higher than that of the anterior.

one sees the peduncle passing inwards and backwards along the outer aspect of the accessory nucleus of the auditory nerve (with which it has fine connections) and the external division of the restiform body towards the lateral angle of the fourth ventricle. On its reaching this angle it becomes forked and one part appears to pass inwards along the floor of the fourth ventricle, while the other part appears either to stop short, or to pass a little way upwards along the side of the ventricle, but owing to the angle which this part of the peduncle makes with the plane of perfectly transverse sections the fibres disappear from view more or less completely on reaching the margin of the ventricle. It is only by careful comparison of a series of sections that their further course can be traced (fig. 1).

With the view of further testing these conflicting views I have made a fresh series of sections in various directions.

Firstly, a series of perfectly transverse antero-posterior sections, at right angles to the plane of the raphé from a fœtus of $6\frac{1}{2}$ months, showed that all the fibres of the flocculus that were myelinated passed inwards along the floor of the ventricle towards the auditory nuclei. None of these fibres apparently passed backwards towards the cerebellum.

Secondly, transverse sections made parallel to the plane of the floor of the fourth ventricle were made to ascertain the angle at which that part of the peduncle which passed inwards formed with the raphe. This angle was ascertained to be about 50° or 55°, the peduncle passing inwards and upwards as a strand of somewhat diverging fibres.

Next, transverse antero-posterior sections were made at an angle of 50° to 55°, with the plane of the raphé, and these confirmed in a very striking manner the results deduced from the comparison with a series of perfectly transverse sections. The peduncle (fig. 2) was seen to sweep round the accessory nucleus and the outer division of the restiform body, and to pass inwards almost, if not entirely, towards the auditory nuclei. Some of its fibres, to all appearance, end in the external nucleus. A larger number, however, pass under the floor of the fourth ventricle and are seen to break up within the internal auditory nucleus.

A few fibres can, I think, be traced into the nucleus of the sixth nerve, and some of them may possibly pass into the grey reticular formation of the same side. The myelination method does not appear to me to permit one to decide whether any of these fibres cross to the formatio reticularis or sixth nucleus of the opposite side.

In perfectly transverse sections made at a slightly higher level than fig. 1 some of the fibres of the flocculus appear to me to bend upwards towards the cells at the lateral angle of the medulla in a position which corresponds to that given by Bechterew as one of the nuclei of the vestibular roots of the auditory nerve. The fibres at this situation appear to me to break up into a fine network, from which again a comparatively small number pass backwards along the lateral wall of the fourth ventricle towards the vermiform lobe. Their manner of termination I have not been able to establish. It appears to me that the strand described by Stscherbak is not connected with the flocculus at all, but passes from the external nucleus of the auditory nerve to the roof nuclei of the cerebellum.

It would thus appear that the flocculus is an important central connection of, certainly, the nuclei of the vestibular root of the auditory nerve, the accessory nucleus of the cochlear root and, almost certainly, of the sixth nucleus. Its connections will no doubt be more fully traced by Marchi's method.

I should like to point out its closely related position to those somewhat rare tumours of the dura mater which arise from the neighbourhood of the internal auditory meatus, and also that the interference with the functions of this lobe caused by pressure upon it may be one of the causes of the vertigo which is associated with tumours in that situation.

THE RESULTS OF EXPERIMENTAL DESTRUCTION OF THE TUBERCLE OF ROLANDO.

BY WILLIAM ALDREN TURNER, M.D.

(The experiments were made in the Neuro-pathological Laboratory, King's College, in association with Prof. Ferrier, F.R.S.)

In the course of an experimental investigation upon the conduction of sensory impressions in the spinal cord and brain, it was considered necessary to destroy, individually, the tubercles upon the posterior surface of the medulla oblongata. The symptoms following lesions of the clavate and cuneate tubercles have been elsewhere described;[1] in this communication the symptomatology of destruction of the *tubercle of Rolando* alone will be detailed.

It was observed that a lesion, thus localised, induced disturbances of sensation in the region of the fifth cranial nerve on one side, and upon the body and limbs on both sides.

It was found that experiments upon this tubercle caused a constant series of objective sensory phenomena; while those on the spinal cord showed that the effects of hemisection were uncertain and various, as indeed recent investigators have already pointed out.

The value of a localised lesion, such as that of the *tuberculum Rolando*, is obvious, in that being situated quite on the lateral aspect of the medulla, it may be destroyed without damage to the tracts passing up the opposite side; on the other hand, it is difficult to perform a hemisection

[1] *Phil. Trans.*, vol. clxxxv., 1894, p. 755.

of the cord without either directly or indirectly injuring structures situated across the middle line.

In the previous series of experiments upon hemisection of the spinal cord (BRAIN, 1891, p. 496), certain facts were noted, which showed that there did not exist entire uniformity in the symptoms following this operation; and others were at that time observed which were difficult to explain, but upon which subsequent, and more extensive, observation has thrown light. At the time referred to, the clip, as a means of testing cutaneous sensibility in animals, was not in general use; during the past few years, however, it has been almost universally adopted, and by some is probably relied upon exclusively, the earlier and more simple means being largely discarded.

Mott's[1] observations upon monkeys also show that considerable variety of symptoms followed this operation; sometimes the animal responded to cutaneous irritation more readily on the paralysed, sometimes on the non-paralysed side; and at others there appeared to be little difference between the two sides.

It would therefore seem from these, and subsequent experiments, that considerable variation exists in monkeys (and probably also in other animals) in the symptoms following hemisection of the spinal cord.

EXPERIMENTS.

Monkeys only were used in the investigation, and strict antiseptic precautions were invariably adopted. In every case chloroform was employed as an anæsthetic.

The tubercle of Rolando was exposed by an incision in the middle line of the neck, removal of the arch of the atlas and incision of the theca vertebralis. It is seen lying external, and somewhat caudal to, the *tuberculum cuneatum* and is easily recognised by its oval shape and greyer superficial appearance. It may be necessary, in order to bring the tubercle fully into view, to tilt the postero-inferior

[1] Mott, *Phil. Trans.*, 1892, vol. clxxxiii., B.p.

margin of the vermis cerebelli. The galvano-caustic knife was used to destroy the tubercle.

In the experiments to be presently recorded, the clip used had a more powerful grip than that now adopted in the laboratory. It cannot therefore be relied upon as a means of testing *slight* defects of tactile sensibility.

EXPERIMENT I.—Mac. rhœsus, April 23, 1892.

After recovery from the immediate effects of the operation, in which the *left* tubercle was destroyed, the monkey presented the following symptoms:—paresis of the left arm and leg, and insensibility to all forms of stimulation of the left cornea, and left side of the face and forehead; with temporary narrowing of the left palpebral fissure and a less prominent state of the left eye-ball.

On the day following the operation the animal was prostrate, and it was difficult to determine with certainty the state of cutaneous sensibility. On the third day, however, recovery had taken place, and the following symptoms were observed: there was no obvious evidence of the presence of tactile sensibility on the left limbs or left side of the body, for the animal paid no attention to gentle touching, ruffling the hair, or dropping water on this side, nor was the left hand or foot withdrawn when dipped into cold water, while a response was at once obtained when the right arm and leg were similarly irritated.

When the *clip* was attached to the left limbs, the monkey struggled and tried to bite, but made no attempt to remove it: on the other hand, when similarly attached to the right side there was no struggling, but the clip was at once pulled off.

Pricking gently the left side of the body, the left limbs and the right side of the face, invariably produced evidence of discomfort, for the monkey would struggle or bite, but no such response was obtained from the right lower limbs or the left side of the face; when the right arm was pricked the monkey occasionally gave some slight sign of sensation by biting or withdrawing the limb, but here it was more difficult to exclude possible vision.

Five days later there was obtained evidence of a sense of touch on the left or paretic limbs. The animal was on several occasions seen to scratch the place on the left arm where it was gently tickled.

The monkey remained in this condition without any very obvious change in the symptoms until June 22, when it was killed under chloroform. The cornea remained throughout anæs-

thetic and did not show any evidence of opacity or ulceration. There was only at the outset a temporary conjunctivitis and slight œdema of the eyelids.

Autopsy.—The lesion involved the tubercle of Rolando throughout its extent, the actual amount of destruction being shown in figs. 1 and 2.

In addition to the lesion of the tubercle, the adjacent structures were involved indirectly by extension of traumatic influences. The structures thus implicated were—the clavate and cuneate tubercles only in their proximal parts, the pyramidal tract as it bends from its decussation into the lateral column of the spinal cord, and the postero-lateral part of the grey reticular formation. The cicatrix, however, was chiefly confined to the gelatinous substance forming the tubercle of Rolando, which was completely destroyed.

EXPERIMENT II.—Mac. sinicus; April 26, 1892.

In this case also the *left* tubercle was destroyed. As a considerable quantity of blood was lost at the time of the operation, the monkey remained weak for four days. On regaining strength on the *fifth* day, the following were observed: a response to tactile impressions was obtained from both sides; either foot or hand was withdrawn from cold water, and an object was grasped when inserted into the palms or the soles. When the clip was applied to the right limbs, it was immediately withdrawn by the monkey, but there was little or no evidence that this was felt as a painful sensation, for there was no struggling or attempts at biting, which invariably occurred when the clip was attached to the left limbs. At times also the clip was localised on this side, more especially when applied to the buttocks and about the root of the tail.

Paresis of the left arm and leg, and anæsthesia of the left face, forehead and cornea existed.

This condition remained practically unchanged during the next fortnight. At this time the paresis had to a large extent passed off. Localisation also of tactile impressions was distinctly present on the left side, although the response was not so active as on the right.

The monkey was killed on June 9, 1892. By this time the paresis had disappeared, with the exception of loss of power of the finer movements of the left hand. The cornea was not now completely anæsthetic, and there was no evidence of any ulceration or opacity.

Autopsy.—The lesion in this case does not appear to have been so complete as in the previous experiment. Owing to some error in preparing the brain for microscopic examination, it has been found impossible to reproduce a photograph of the lesion; but from a study of the sections obtained, it was seen that the structures implicated were: the tubercle of Rolando, the top of the left crossed pyramidal tract, the adjacent cuneate tubercle and the dorsal part of the left grey reticular formation.

EXPERIMENT III.—"Sooty" monkey, male; October 29, 1892. Destruction of the *right* tubercle of Rolando.

After recovery from the operation the monkey presented considerable disturbance of equilibrium; there was a tendency to roll towards the right side, and it was unable to sit up without support. There was paresis of the right arm and leg; the right pupil was smaller than the left, and the right cornea and side of the face were anæsthetic. Gentle cutaneous irritation of the left limbs and left side of the body always caused the monkey to scratch the part so irritated, but no evidence of the presence of tactile sensibility was obtained on the right side. Pinching, pricking and the clip were invariably localised on the left side but not on the right.

The above was the only observation made upon this monkey. From the fact that it was suffering from diarrhœa at the time of the experiment, it did not long survive the operation.

Subsequent examination of the site of the lesion showed that the tubercle of Rolando was destroyed, and as in the previous cases the pyramidal tract, the adjacent portions of the grey reticular formation and *funiculus cuneatus* were slightly implicated.

EXPERIMENT IV.—Mac. rhœsus; November 22, 1892.

In this case the *right* tubercle was destroyed. On the day after the operation the monkey presented the following symptoms: paresis of the right arm and leg; there was no obvious evidence of the presence of tactile sensibility on the right limbs, as tested by gentle ruffling of the hair and the dropping of water; while similar irritation was at once localised on the left side by scratching. It bore with equanimity pricking on the left side, which caused active manifestations of feeling on the right. When the clip was applied to the right foot, struggling at once ensued; when attached to the left there was no struggling, but the animal at once withdrew it; it never attempted to withdraw the clip

from the *right* foot. There was anæsthesia of the right cornea and right side of the face, and the right pupil was smaller than the left.

Two days later there was still no clear evidence that the monkey was aware of dropping water, or gentle rubbing of the skin of the right arm or leg. As evidence of analgesia of the left side may be mentioned the fact, that the monkey leant this side against the hot plate of the stove without apparent discomfort.

At the end of the week, however, there was no doubt that the monkey was aware of the gentlest touch on the right leg and right side of the body; it moved away from the source of irritation, but it did not localise by scratching, although it looked round to the place of irritation.

Six weeks after the operation the monkey was in good health. Although the right arm was still paretic, the leg had, to a great extent, recovered. It bore on the left limbs the pressure of forceps, which occasioned struggling and discomfort when applied to the right. The right cornea remained clear throughout, but later on its sensibility and that of the right side of the face was merely defective as compared with the opposite side. The monkey was killed on January 23, 1893, without any further change in the symptoms having occurred.

Autopsy.—The lesion, in this case, was practically the same as that in the first experiment. The amount of destruction has been reproduced in figures 3 and 4. At the lower part (distal) of the lesion, the only structure destroyed was the gelatinous substance of Rolando; while in the upper part, in addition to this, there were involved, and partly destroyed, the cuneate tubercle and the adjacent dorsal part of the grey reticular formation. But here again, the cicatrix produced by the cautery occupied the position of the *tuberculum Rolando*.

Remarks.

The effects of localised destruction of the tubercle of Rolando, as observed in these experiments, may be divided into two sets; firstly, defects of sensibility over the distribution of the fifth cranial nerve on the side of the lesion; and secondly, alterations in the sensibility of the body and limbs on both sides.

(1) The phenomena over the distribution of the trigeminal nerve consisted in defect or abolition of all forms of sensation in the cornea, the skin and the mucous membranes supplied by that nerve. The loss of sensibility thus obtained, was due to the destruction of the so-called "ascending" trigeminal root, where it lies upon, and forms the superficial white stratum of, the Rolandic tubercle. It was specially observed in those cases, that, although the cornea remained anæsthetic throughout the life of the animal after the operation, in no case were "trophic" changes noted either in the form of corneal opacity, ulceration or ophthalmitis. This subject, however, has been submitted to more extensive experimental investigation, and brief reference has been already made to it.[1] The details of these experiments will be given in another communication.

Among other and minor symptoms noticed, were narrowing of the pupil on the side of the lesion, and in one experiment a very decided narrowing of the palpebral fissure on the same side. The existence of pupil-dilating fibres in the ophthalmic branch and sensory trunk of the trigeminus was thus confirmed, and their presence in the so-called "ascending" root has been established by these experiments.

(2) Coming now to the more important physiological question concerning the sensory phenomena in the body and limbs after this lesion, it will be noticed that the symptoms present a marked degree of uniformity.

The general statement may be made that, *on the side of the lesion*, there was temporarily after the operation no apparent evidence of the presence of a sense of touch, or of localisation, although in one case (Experiment II.), a sense of contact appeared to be present, but defective; while the sense of pain, as such, was in every instance retained. In the second experiment, however, the symptoms were not observed until the fifth day, by which time some recovery had probably taken place. *On the opposite side*, tactile impressions were invariably localised, while there was loss or defect of the sense of pain, as such.

[1] Ferrier, BRAIN, 1894, p. 1.

although the site of irritation, whether this was effected by pricking, or by the application of the clip, was accurately localised. On the side of lesion there was weakness of the voluntary movements of the arm and leg, which, on subsequent examination, was found to be due to injury to the fibres of the crossed pyramidal tract under the tubercle of Rolando (*vide* figures).

In order to compare these results with those of hemisection of the spinal cord by the same methods, the following two experiments were made.

EXPERIMENT V.—Section of the *left* side of the spinal cord. Mac. cynomolgus; June 11, 1892.

On recovery from the anæsthetic, the monkey was found to be paraplegic and to pay no attention to sensory impressions of any sort on either lower limb. There was no general shock, as it was otherwise active and took food well. This condition remained until the *third* day after the operation. When tested on this occasion, the monkey accurately localised the clip, pricking and gentle scratching of the skin of the *right* lower limb and right side of abdomen, but it gave no sign of pain or discomfort. It paid absolutely no attention to any form of irritation on the left lower limb. Both legs remained completely paralysed. The temperature of both soles was nearly equal: left sole 89·5° F., right sole, 90·5° F. The knee-jerks were equal and apparently normal. At the end of a week slight movements of flexion at the right hip-joint were detected, but the state of sensation remained as before. Although the animal invariably localised, where it was touched or pricked on the right lower limb, there was no evidence by struggling or otherwise that the latter was felt as painful. The left lower limb remained completely paralysed, analgesic and anæsthetic until the death of the animal six weeks after the operation.

Autopsy.—The lesion was made between the sixth and seventh dorsal nerves. Sections taken from the seat of lesion after hardening in Müller's fluid and staining by the Weigert-Pal method showed that the cicatrix extended across the middle plane. The whole of the left side of the spinal cord was divided and the incision filled with cicatricial tissue, which extended across so as to obliterate the

posterior column of the opposite side, and, to some extent, the posterior horn of grey matter. The opposite lateral and anterior columns and the anterior horn were unaffected (fig. 5).

EXPERIMENT VI.—Section of the *left* side of the spinal cord in the upper dorsal region. Mac. sinicus; January 24, 1893.

On the day after the operation the monkey was submitted to careful testing, and the following facts were observed:—There was complete paralysis of the left leg; the repeated prick of a needle and the application of the clip were accurately localised upon the *right* leg, but no struggling or other evidence of the appreciation of painful sensation was observed, such as invariably happened when the *left* leg was pricked or the clip applied. On this side struggling followed pricking and the clip, but there was no attempt at localisation on the left lower limb. There was no clear evidence of a sense of contact on either leg, other than that already noted. Tactile impressions were readily detected on the arms.

One week after the operation, however, definite evidence was obtained of a sense of touch on the *right* foot, for when gently touched with the finger or when blown upon, the foot was withdrawn, and at times the monkey would scratch the place so irritated.

Three weeks after the operation the *left* lower limb remained completely paralysed, nor was there any evidence of the presence of a sense of touch upon this limb. Pricking and the clip gave active manifestations of feeling, but without localisation, while on the *right* side, although impressions both of touch and pain were localised, there were no signs that the latter was felt as such.

Autopsy.—The lesion itself was an almost exact hemisection, and is shown in fig. 6. With the exception of the mesial part of the anterior column, the whole of the left side of the cord was formed of the cicatricial tissue. The secondary degenerations also indicated an almost complete hemisection, for with the exception of a few scattered black spots (by Marchi's method) in the antero-lateral superficies of the right side, the resulting descending degeneration was limited to the lateral and antero-lateral columns on the side of the lesion.

Closer analysis of these two experiments shows certain interesting features. The symptoms immediately following the operation in Experiment V. were those of a complete transverse section of the spinal cord, both as regards motion, sensation, and the knee-jerks. On the third day recovery of tactile sensibility took place on the side opposite the lesion, but the state of motion remained defective for a longer time. To look carefully at the actual amount of destruction of the cord which had taken place, it will be seen that, in addition to the hemisection of the left side, the whole posterior column and the greater part of the posterior horn of grey matter on the right side, has been replaced by cicatricial tissue; while of the remainder of the grey matter, the anterior horn and adjacent tissue remains unaffected. So far, therefore, as the posterior region of the cord is concerned, the lesion is, to all intent, bi-lateral. This no doubt explains the bi-lateral anæsthesia and analgesia, which was temporarily observed. The early return of the sense of touch on the side partially destroyed, indicates that the tracts subserving touch may have a more general distribution throughout the grey matter than those of pain, in which case a greater amount of destruction is necessary to abolish tactile sensibility—a view which may explain the dissociated loss of painful sensibility in syringomyelia.

The symptoms observed in these two cases, especially Experiment VI., differed to some extent from those previously recorded after hemisection of the spinal cord (BRAIN, *op. cit.*), as well as from those observed by Mott[1] after a similar operation.

They emphasise what had been previously observed, and what was very apparent in Mott's experiments, viz., the removal of the clip only from the side opposite the lesion. It is difficult to state what form of sensation the application of the clip conveys, but it has been found from an extensive series of observations upon the production of sensory disturbances in monkeys, that the removal of the clip goes *pari passu* with the existence, but especially the

[1] Mott, *Phil. Trans.*, *op. supra cit.*

localisation, of tactile sensibility, ascertained by other methods, whatever be the state of the motor functions. The removal of the clip from a paralysed leg was well illustrated in Experiment V., in which, as a result of hemisection, both legs were paralysed. The monkey, however, accurately localised the clip, on the side opposite the lesion.

It is necessary to point out, that although the clip was removed from the limbs on the side opposite the lesion, both after hemisection and after destruction of the tubercle of Rolando, there was no evidence of a sensation of pain as such. Neither by struggling nor otherwise did the animal give any evidence of this; while with the exception of the complex Experiment V., the application of the clip invariably gave rise to struggling on the side of the lesion. In both series of experiments the evidence bearing upon the loss of the sense of pain on the side opposite the lesion was most convincing. The sense of touch, however, appears to be differently conducted. In none of the experiments was there any clear evidence, at least temporarily, of the presence of a sense of touch on the side of lesion, while it was invariably shown to be present on the opposite side.

There are few experiments upon the region of the Rolandic tubercle, a record of which I have been able to obtain. Three are recorded by Herzen and Loewenthal.[1] Their experiments were performed on cats, but instead of destroying the tubercle of Rolando, they made an incision through the posterior column and the dorsal part of the lateral column on one side, at the junction of the spinal cord and medulla oblongata. The consequent disturbances of sensation do not seem to have been very minutely tested, but the general result is in harmony with those recorded in this paper. For example, in the first experiment, one month after the operation, the animal bore the contact of a piece of ice on the paws on the side of the lesion without any sign of sensation, but at once withdrew them when ice was applied to the opposite side.

In the second experiment sensation appeared to be less active on the side of the lesion than on the opposite side.

[1] Herzen and Loewenthal, *Arch. de Physiol.*, 1886, p. 260.

In the third case, a more energetic response followed pinching the toes of the paw on the side of the lesion than on the opposite side; while, as in the first case, one month after the operation, the limbs on the side of the lesion could be immersed in ice water without the slightest sign of sensation, while those on the opposite side were withdrawn on the first contact with the water. Further, no notice was taken of touching the hind limb on the side of lesion, while that on the opposite side was at once moved.

From this it appears that tactile sensibility, chiefly ascertained by the contact of ice, was abolished on the side of the lesion; painful sensation, on the other hand, tested by pinching, was defective on the side opposite the lesion.

Bechterew[1] also records an experiment which he performed upon a dog in this region. By passing a knife through the occipito-atloid foramen, he was able to divide the following structures on the right side — the greater part of Burdach's column, the fibres of the posterior decussation, the outer part of the reticular formation, as well as the gelatinous substance of Rolando and the ascending trigeminal root. As a result of this lesion he found, that the right side of the face, mouth, tongue, and the right cornea were anæsthetic and analgesic; and that the tactile sensibility on the right limbs was distinctly defective compared with the left, the sense of position of these limbs also was lost. On the left limbs the animal responded to the slightest cutaneous irritation, but made no response to painful impressions. The motor power of the limbs was not materially altered.

I have been unable to find the record of any case of disease or injury in man at this locality, which was followed by a *post-mortem* examination. There are two cases, however, in which the symptoms point to a lesion at or about the junction of the spinal cord and medulla oblongata.

(1) *Bechterew's Case.*[2]—The symptoms were observed one year after a bullet wound was inflicted on the left side at the level of the atlas; on the *right* side there was anæsthesia of the face, paralysis, eventually becoming merely weakness,

[1] Bechterew, *Neurol. Centralb.*, 1894, p. 298.
[2] Bechterew, *Neurol. Centralb.*, 1893, p. 457.

of the right arm and leg, dulling of the sense of touch and complete palsy of the "muscle-sense." On the *left* side there was paresis of the arm and leg, which eventually disappeared, loss of the senses of pain and temperature, dulling of the sense of touch, but retention of the "muscle-sense."

In this case, as the motor symptoms show, the lesion was not completely unilateral, a fact which may account for the dulling of tactile sensibility on both sides. The condition of painful sensibility on the right side is not stated; nor is a definition given of the term "muscle-sense." If it may be interpreted as the sense of position, then its retention on the side on which tactile sensibility was least affected, and associated with loss of painful sensibility, is in close harmony with the recorded experiments upon the Rolandic tubercle.

(2) *Steiglitz's Case.*[1]—The *right* side of the face and right cornea were completely anæsthetic; the right side of the body, though paralysed, was sensible to the slightest touch, the pressure of the bedclothes being almost painful; but the sense of position of the limbs was uncertain. On the *left* side there was anæsthesia to touch, pain and temperature, but there was no ataxy of the left limbs.

In this case the association of anæsthesia of the face and cornea, with altered sensibility of the body and limbs, suggest a lesion above the second cervical nerve root, and in close proximity to the tubercle of Rolando. From the complete decussation of the senses of touch and pain, it is more probable, however, that the lesion is higher up in the medulla oblongata.

The original experiments recorded here, taken in association with those of Herzen, Loewenthal and Bechterew, show that at the medullary-spinal junction the paths conducting the sensations of touch and pain are represented on both sides; the path for painful sensibility being crossed, and that for touch both crossed and uncrossed.

It would appear, however, that lower down in the spinal cord there is no absolutely constant distribution of the

[1] Steiglitz, *Neurol. Centralb.*, 1893, p. 145.

fibres entering by the posterior nerve roots. Sometimes it was found experimentally that all forms of sensation were abolished on the side opposite the lesion, or again, that one form (touch) was abolished on the side of lesion, and another (pain) on the side opposite.

Coming, however, to the spinal-medullary junction, we find a more uniform method in the arrangement of the sensory tracts. At this level the path of painful impressions has entirely decussated, while those of touch are passing up more or less on both sides. In course of time after the operation, some recovery takes place, and we find the animal, whose Rolandic tubercle has been destroyed, again localising cutaneous impressions within a few weeks after the operation.

But if we now consider what are the probable paths of sensation, we find valuable evidence from the experiments on the Rolandic tubercle, taken in conjunction with Experiment V.

In the first series, the lesion was, for all practical purposes, limited to the gelatinous substance and posterior horn, and the symptoms were, loss of touch on the same side and loss of pain on the opposite.

In Experiment V. both posterior columns, one posterior horn completely and the opposite horn *partially*, were destroyed, and the symptoms were complete anæsthesia and analgesia on both limbs, but after the third day sensibility to touch returned on the side on which the posterior horn was only partially involved.

One may therefore conclude that the posterior horns contain the fibres, which subserve the cutaneous sensibility, rather than the posterior columns, which in the spinal experiment were both completely destroyed, and in the tubercle experiments only partially involved, and that by secondary traumatic rather than direct influence.

An examination of the figures shows that the gelatinous substance of Rolando, with the subjacent posterior horn, has been chiefly destroyed, although neighbouring structures have been, to a slight extent, secondarily involved. From the results of the experiments, it would appear that the

gelatinous and spongy substances of the posterior horn may be of greater importance in the conduction of sensory impressions than has hitherto been suspected.

When examined histologically by the Golgi method, the gelatinous and spongy substances of the tubercle of Rolando are found to contain a large number of fine nerve fibres and their end-tufts, which the observations of Kölliker[1] and Held[2] have shown to be, in the medulla, the axis-cylinder processes and collaterals of the cells of the Gasserian ganglion. In the spinal cord a similar structure is observed, being in this case the terminal nucleus of certain of the posterior root fibres. Situated in the substantia spongiosa, in close relation to the end-tufts, are large multipolar nerve cells, whose axis-cylinder processes pass into the tegmentum medullæ.

It has also been shown that section of the sensory trunk of the fifth nerve proximal to the Gasserian ganglion, or of the so-called "ascending" trigeminal root in the medulla, was followed by degeneration and atrophy of the fibres, which pass through the gelatinous substance and of the network which these fibres form in the spongy substance of the posterior horn. No atrophy of the cells, on the other hand, was detected in this situation.[3]

What the further course of the sensory trigeminal fibres may be, is more difficult to state. Owing to the interruption of degeneration, which occurs in the substantia spongiosa, no direct observations by this method have been recorded; but the observations of Kölliker and Held point to a passage across the raphé in the medulla and pons of the axis-cylinder processes of the cells of the substantia spongiosa.

This crossed relation of the sensory trigeminal fibres, as well as of the sensory fibres from the body and limbs, was corroborated by an experiment, where, after hemisection of the tegmentum pontis, anæsthesia of the opposite half of the body was detected.

As elsewhere shown (*Phil. Trans., op. cit.*), destruction of the nuclei of Goll and Burdach was not followed by any

[1] Kölliker, *Handb. d. Gewebelehre*, 1893.
[2] Held, *Arch. f. Anat. und Physiol. Anat. Abtheil.*, 1893.
[3] *Phil. Trans.*, vol. clxxxv., 1894, p. 763.

appreciable defect of common sensibility in the body or limbs; and Mott (BRAIN, 1895, p. 11) has more recently stated, that it was impossible to make out anything definite regarding sensation after severence of the fibres leaving these nuclei. Now as these nuclei give origin to the fibres forming the fillet of the opposite side, it is impossible to support the view that the fillet is the tract of ordinary cutaneous sensation, at all events in the lower (distal) portions of the medulla oblongata. Moreover, there are cases on record of hæmorrhage into, or softening of, the tegmentum pontis in which the fillet was intact, but which were accompanied by anæsthesia of the opposite side. Added to this is the more important fact, which the experiments here recorded have brought out, that destruction of an entirely different region, viz., the posterior horn and gelatinous substance of Rolando, is followed by characteristic sensory disturbances on both sides of the body.

The experimental evidence in this paper is in favour of the view suggested by Tooth[1] on anatomical grounds, that the gelatinous substance and the adjacent part of the posterior horn are chiefly concerned in the transmission of sensory impressions.

[1] Tooth, *Journal of Physiol.*, Supplement, 1892, p. 173.

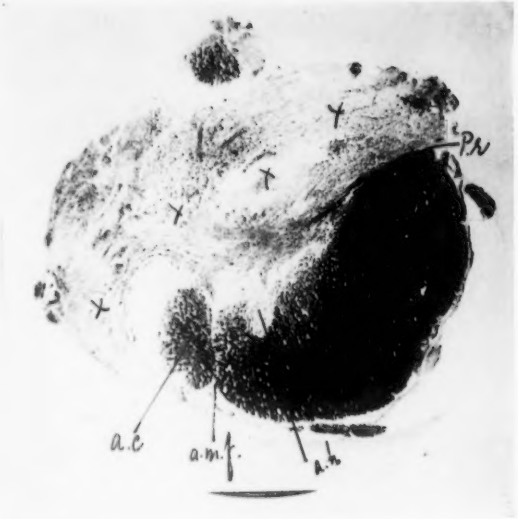


Fig. 5.

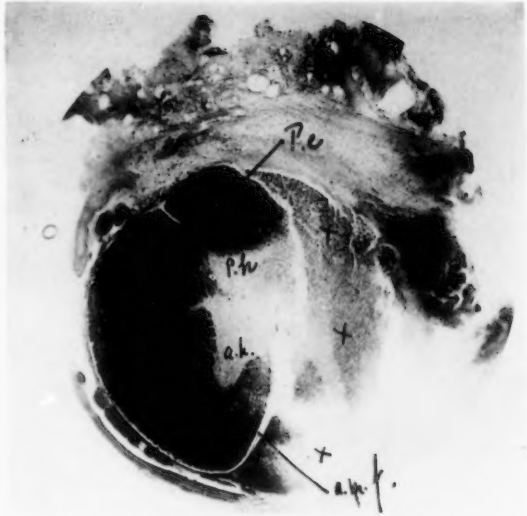


Fig. 6.

Collotype. J. J. Briginshaw, Sise Lane, 35d, Queen Victoria Street, E.C.

Fig. 3.

Collotype. J. J. Briginshaw, Size Lane, 35d, Queen Victoria Street, E.C.

Fig. 4.

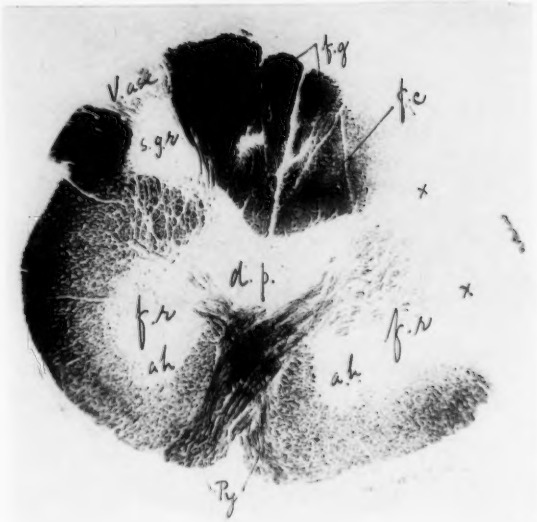
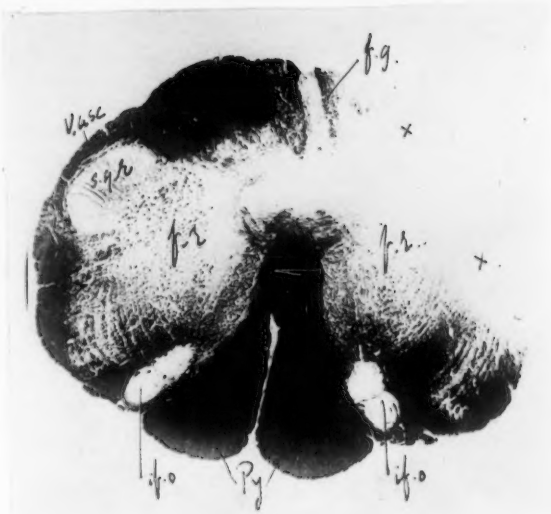


Fig. 1.



Collotype. J. J. Briginshaw, Sise Lane, 35d, Queen Victoria Street, E.C.

Fig. 2.

EXPLANATION OF THE FIGURES.

FIGS. 1 and 2 illustrate the lesion in Experiment I.

xx. indicate the position of the lesion.
f.g. funiculus gracilis.
f.c. funiculus cuneatus.
s.g.r. gelatinous substance of Rolando.
a.h. anterior horn.
d.p. pyramidal decussation.
f.r. reticular formation.
if.o. inferior olivary body.
Py. pyramids.
V. asc. ascending root of trigeminus.

FIGS. 3 and 4 illustrate the lesion in Experiment IV.

xx. indicate the position of the lesion.
The lettering is the same as in figs. 1 and 2.

FIG. 5 is a section through the lesion from Experiment V. It will be seen that the whole of one side of the spinal cord, with the exception of the anterior column, is replaced by cicatricial tissue, which extends across to the posterior horn of the opposite side, this also being partially implicated.

FIG. 6 represents the extent of the lesion in Experiment VI.

a.h. anterior horn.
p.h. posterior horn.
P.c. posterior column.
a.m.f. anterior median fissure.
xxx. cicatrix.

Note.—In the figures representing the lesion of the Rolandic tubercle the outline of the cicatrix has not been depicted. This is due to the fact that the sections having been stained by the Weigert-Pal method, the fibrous tissue of the cicatrix was not sufficiently dense to retain any colour.

THE MORBID ANATOMY OF A CASE OF INFANTILE PARALYSIS.

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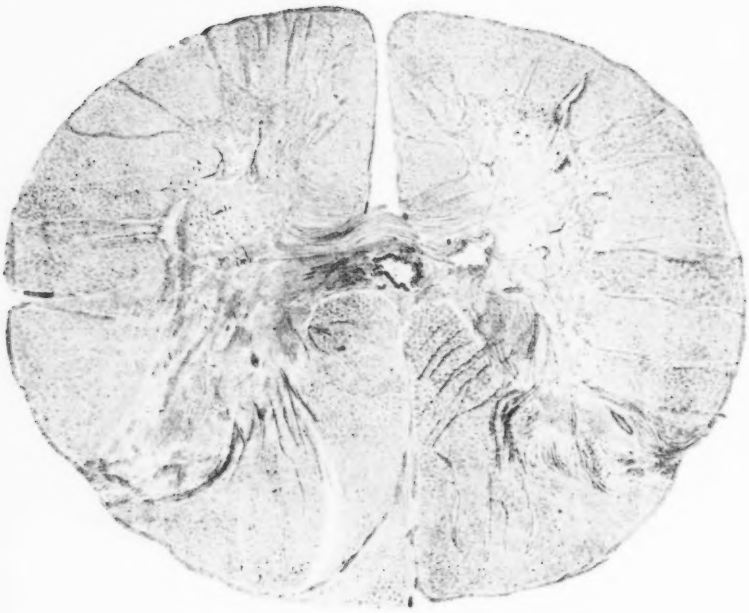
(*From the Laboratory of the Leeds General Infirmary.*)

THE following is an outline account of the case for the notes of which I am greatly indebted to Dr. Chadwick and Mr. Brown, under whose care the patient was.

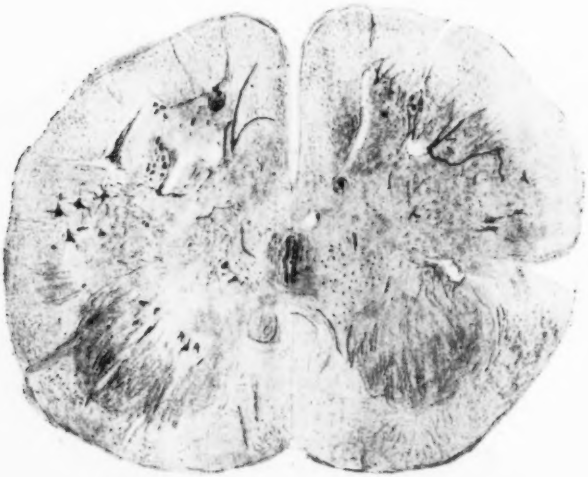
A girl, aged 6 years, was suddenly seized, nine months before admission, with fever, nausea and vomiting. Paralysis appeared on the second day, involving all four extremities. The fever ceased in nine days. The urine and fæces were passed into the bed for three weeks. The arms began to recover three weeks after the onset. On admission there was almost complete paraplegia with contracture in the calf muscles. The right arm was weak. Hardly any wasting was noted. The plantar and patellar reflexes were absent, the abdominal present. The reaction to faradism was absent, that to galvanism weak. K.C.C. = A.C.C. Sensation was intact. Later some improvement occurred in the legs, apparently under massage. Death took place about eleven months after the onset of the disease from independent causes.

At the necropsy there was slight diffuse nephritis with marked fatty changes in the glomeruli of the kidney. There was no obvious naked eye changes in the cord or brain. Unfortunately, I omitted to obtain any of the peripheral nerves or muscles.

A word may perhaps be said here in respect to methods. The rapid method of hardening in Müller's fluid in a hot oven at body temperature was adopted. In two and a-half



General View.—(*a*) Lumbar region. × 2 in.



General View.—(*b*) Lower sacral region. × 2 in.

days the material was taken out and put through alcohol of increasing strengths. In five days it was quite ready for embedding in paraffin. Sections were cut with the rocking microtome and were stained after Weigert's method, in aniline blue black, logwood and eosin, borax carmine or picro-carmine and logwood. A few sections were stained after Pal's method, and a few without much result in Rosin's triple stain.

Numerous sections were examined from both ends of the spinal nerve segments, longitudinal and sagittal sections through the horns being taken in the intervening parts. Sections were also examined from the M.O., pons, crura, internal capsule and motor cortex, but no lesions were found in these situations.

It may save space to summarise the morbid changes found in the various elements such as have been ascertained to exist in considerably over 200 stained specimens carefully examined and systematically noted.

Ganglion Cells.—The ordinary changes in these cells are readily seen. Alteration in and disappearance of the processes, as well as shrinking of the cell into an amorphous looking mass were observed. An indistinctness in the outline and a breaking up of the cell were occasionally seen. Only very exceptionally were round cells found in the perigangliar space. Spaces where former ganglion cells had been were fairly numerous. A stippling of the general protoplasm of the cell was only obvious at times. There was no distinct evidence obtained of pigmentation or calcification of the cell. The changes in the nucleus and nucleolus were very interesting. It is often said that the nucleus and nucleolus disappear early, but there would appear to be circumstances under which these structures are more resistant to such changes. Shapeless masses of protoplasm were here sometimes recognised as the remains of ganglion cells solely by the presence of a distinct circular nucleus often with nucleolus obvious only on careful focussing. Some little time ago I was much struck when studying sections from a dog's cord with a spontaneous acute myelitis of five days' duration, to find that the nucleus of these cells could

at times be seen almost shorn of all trace of surrounding protoplasm.

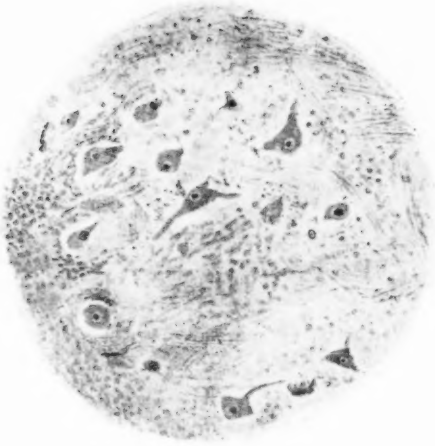
The above-named changes in the ganglion cells were best studied in the material from this case in the upper cervical and lower dorsal regions, and especially perhaps in Clarke's column (fig. 1). Groups of minute and perfectly formed ganglion cells were here and there seen chiefly in the parts corresponding to the lateral horns. Kohnstamm (*Zeitschr. f. klin. Med.*, 1893) has specially drawn attention to these cells. Once, and perhaps twice, a ganglion cell was seen to have two nuclei. In no part of the cord could the destruction of ganglion cells be said to be limited to one or more groups in the sense insisted upon by v. Kahlden.

The *nerve fibrils* in the grey matter were greatly changed. They had often entirely disappeared. When present they were mostly reduced to fine lines, and frequently they were distinctly varicose (fig. 2). These changes were readily studied in the anterior commissure. This fact is not so much to be wondered at if the median and hinder group of ganglion cells are to be looked upon as commissural cells (Lenhossek), for these latter were greatly involved throughout.

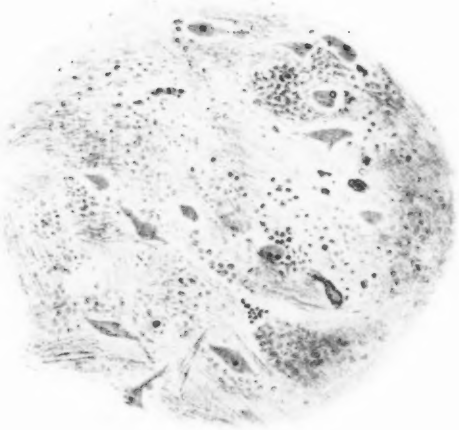
The *anterior nerve roots* as they passed out through the white matter seemed to be less well represented than usual, the individual fibres being fewer in numbers and thinner. Only in a few specimens did they appear to be degenerate. The posterior roots were intact.

The *neuroglia* of the grey matter was considerably altered, chiefly in the direction of overgrowth and infiltration. In many sections taken from the lumbar and cervical enlargements translucent areas were visible in the grey matter. These were unquestionably due to the destruction of the numerous nerve fibrils and ganglion cells. If such a translucent patch was examined under a high power, the overgrowth in the neuroglial element became obvious. It consisted of a fairly open rich network of thick fibres, with the presence here and there of enormous spider cells (fig. 3), whose processes could be distinctly seen to form this neuroglial network. In parts of the cervical region these

FIG. 1.



(*a*)



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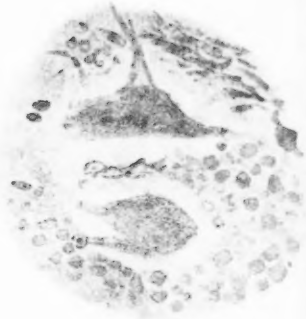

(*c*)

Ganglion cells healthy and in various stages of degeneration in Clarke's columns. The cellular infiltration is very well marked. Upper dorsal region.

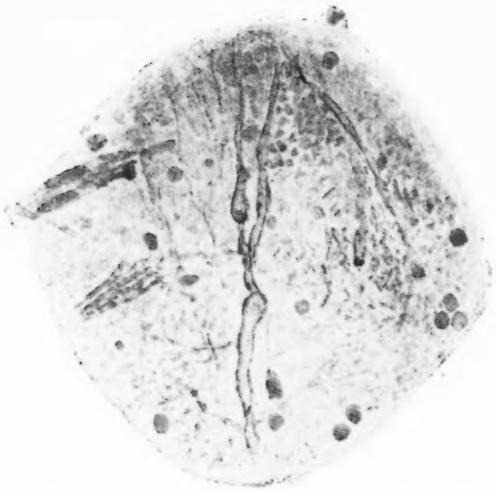

FIG. 2.
Varicose and wasted nerve fibrils from the upper lumbar region. × ½.

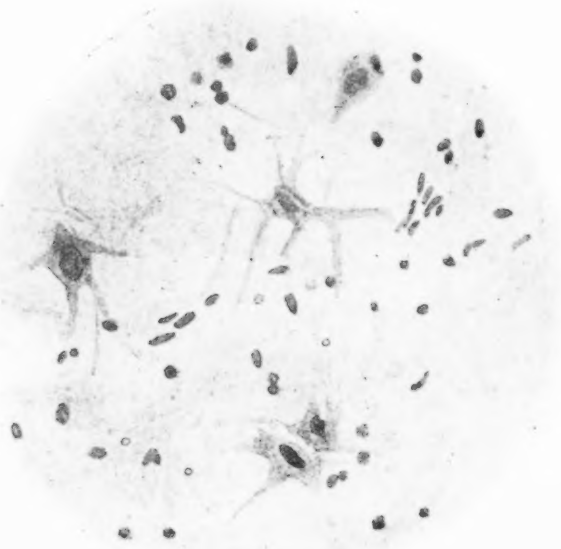

FIG. 3.
Enormous spider cells (lumbar enlargement). × ½.

spider cells were present in great numbers. In Edinger's admirable *Vorlesungen*, 1893, the opinion is still expressed that these spider cells are really thin cell plates and that fibres only appear to arise out of the cells because they lie in close relation to them. That the fibres in question do arise from the cells in these specimens there can be no manner of doubt, and indeed, researches with Golgi's method seem to have set this question definitely at rest. (Lenhossek, *Der feinere bau des Nervensystems*, 1893.)

The *central canal* is pervious almost throughout the cord. In its neighbourhood are a number of large epithelial-looking cells clearly derived from the cells lining the central canal.

There are two further pathological processes in the neuroglia now to be considered, namely, the increased vascularity and cellular infiltration.

Vascularity.—In many parts of the cord there is obviously greatly increased vascularity. Here a few remarks may be made on the vascular supply of the cord especially as the vascular and interstitial origin of the disease is adopted here. It consists of central and peripheral components. The central artery penetrates into the anterior fissure at the bottom of which it divides. From this central supply Clarke's column is provided. These branches of the central artery can fairly readily be made out if a number of sections are examined.

The peripheral supply is chiefly limited to the white matter. It is very obvious, however, that the outermost parts of the grey matter also receive blood from the peripheral as well as the central arteries. In some of the sections such a branch can be seen to penetrate the anterior horn. As will be pointed out later, some of the advanced perivascular changes lie in a zone in which the central and peripheral arteries would appear to meet. Any distinct disease of the arterial wall itself I have not been able to make out except in a few doubtful instances. Once a capillary appeared varicose. The lymphatic sheath, however, was frequently found crowded with round cells.

Cellular Infiltration.—In most parts of the cord, and most marked in the mid-cervical region, there was very

considerable cellular increase. These cells may roughly be divided, as has been done by Dauber and others, into (1) round cells with a very large nucleus, and (2) cells with many processes, namely Deiter's or spider cells. These latter would appear to vary very considerably in size. The cell infiltration is distinctly of two kinds: (1) patchy, (2) general. The patchy infiltration is frequently seen to be distinctly perivascular. The original specimen from which the drawing (fig. 4) comes, strangely reminds one of a recent infiltration. In another longitudinal section from the lumbar enlargement (fig. 5) the change would appear of a more chronic character, the neuroglia round these vessels being unquestionably considerably altered.

There is yet another infiltration, if it may be so called, round about the central canal and penetrating at times deeply into the surrounding parts. It consists of very large, often irregular, cells with no distinct processes. They have all the appearances of being derived from the cells lining the central canal. Since these cells have a similar origin to Deiter's cells, their increase and multiplication is hardly to be wondered at (see Lenhossek).

White Matter.—The changes here are much less obvious but yet distinctly present. In the neighbourhood of the diseased horns the neuroglial element is denser and its cells more numerous than usual. In one specimen an unmistakeable patch of round-celled infiltration was seen, and in another logwood eosin preparation a curious staining which looked exactly like the remains of an old hæmorrhage. No corpora amylacea were present. In longitudinal sections (logwood and eosin) the cellular increase could be distinctly made out. Similar sections stained according to Weigert's method showed slight changes in the myelin of the nerve fibres. In a few specimens in which the pia mater and subarachnoid tissue remained, no evidence of any inflammatory change was forthcoming. This is noted, as some observers seem to have found such a lesion present (Goldscheider).

As regards the distribution of the lesion, it extended undoubtedly throughout the whole length of the cord. It

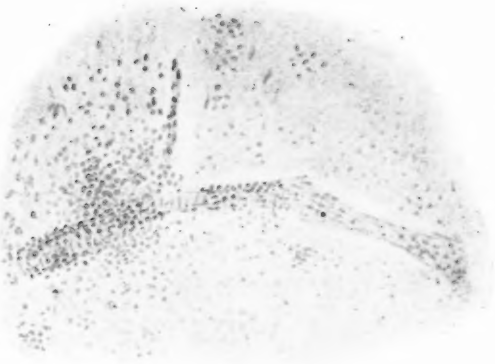

FIG. 4.
Perivascular infiltration (lumbar enlargement) about a vessel quite in the periphery of the grey matter. × $\frac{1}{4}$.

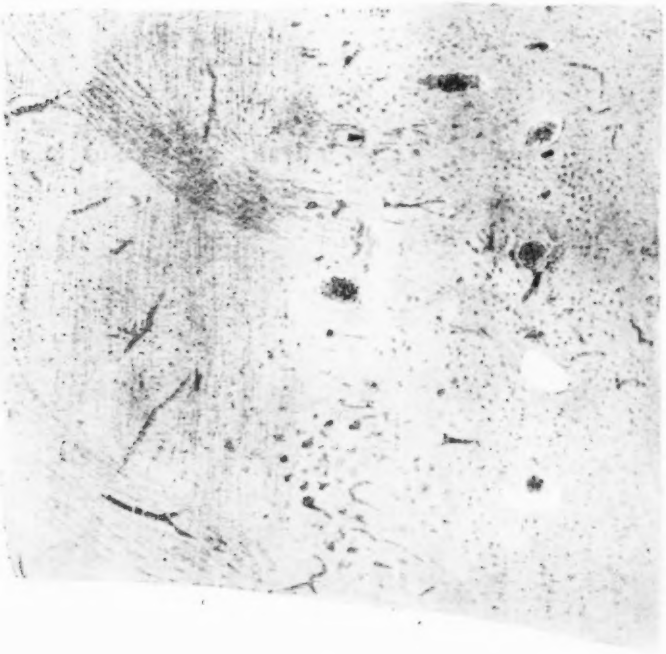

FIG. 5.
Longitudinal section, showing cellular infiltration and perivascular changes.

was present in the lowest sacral segments and reached as high as the decussation of the pyramids. The lesion was unquestionably intense in the *lowest* part of the cervical region (C6 to D1). It gradually diminished in an upward direction. It was also less intense in the upper dorsal region to become more marked in the mid-dorsal region and less pronounced in the lower dorsal cord. In the lumbar region it was very intense, especially about L 2, 3 and 4. Below L 5 it tended to become less marked, but did not altogether disappear even in the lowest sacral segments. It was somewhat less marked, especially in the cervical enlargement, on one side than on the other.

It is proposed briefly to attempt an explanation, however inadequate, of the clinical features of the case, by the morbid lesions found.

The paralysis in the lower limbs is amply accounted for by the disease in the lumbar cord. The improvement noted in the legs in the tenth or eleventh month, so that the child could just lift them from the bed, is curious. In one longitudinal section, however, from the upper lumbar enlargement quite a number of healthy ganglion cells were found. In no other section from this region was a similar appearance noted. It is almost surprising that there was any power at all in the right arm, and yet the patient could move the fingers of the corresponding hand. The amount of power possessed by the left arm is also remarkable, for the child could feed itself with this left arm. It would be a hardihood to attempt to explain the above noted improvement by a regeneration of ganglion cells. Yet those small and perfect ganglion cells noted above suggested such a possibility. Tumours have been found in the central nervous system composed in great measure of ganglion cells, showing that the division and multiplication of these cells is sometimes possible.

The absence of wasting was a striking fact; it was noted clinically, and in the pathological report. The absence of the plantar reflex could sufficiently be accounted for by the disease in the sacral segments, and the absent knee jerk by the intense disease in the lumbar enlargement. The

abdominal reflex was present, and the disease was certainly less intense in the corresponding part of the cord (lower dorsal). Unfortunately there is no note about the other reflexes. The epigastric reflex should have been absent on account of the marked lesion in the mid-dorsal region.

The question of the nature of the lesion in infantile paralysis has been so fully debated of recent times, and the disease so clearly shown to be of vascular and interstitial origin—that is, the changes in the neuroglia including vessels, are primary, those in the nerve cells secondary—that it is unnecessary to discuss it at length. Goldscheider (*Zeitschr. f. klin. Med.*, 1893) and even more specially Siemerling (*Arch. f. Psychiatrie*, xxvi. Bd. i., 1894) have strongly advocated this view. v. Kahlden (*Ziegler's Beiträge*, 1893) has, however, vigorously supported Charcot's opinion that the disease starts in the nerve cells, the changes in the neuroglia being secondary only. Siemerling's rejoinder seemed unanswerable. v. Kahlden has again quite recently returned to the subject in the *Centralbl. f. allg. Pathol.*, Sept., 1894 (Epitome, *Brit. Med. Jour.*, Oct. 20, 1894, par. 292) in a review of recent writings upon this question. He discounts, perhaps hardly pertinently, some of the cases which have been brought forward in favour of the interstitial and vascular origin of the disease, by treating them as atypical cases.

Most of the cases upon which the above authors have based their conclusions are of very recent origin. In the old cases the changes are so advanced as almost to preclude an opinion as to the starting point of the disease. The case recorded above stands in a middle group between these two classes of cases. The changes seem to be entirely in favour of the disease being primary in the neuroglia including the arteries. The infiltration, and especially its perivascular localisation, may be here especially referred to. On the other hand, there is no definite localisation of the disease to any one or more groups of ganglion cells. The overgrowth in the neuroglia, including the spider cells, is certainly striking. This may be due to the irritation of the poisonous products (toxines), escaping from the blood

according to the infective theory of the disease, and this view certainly appears to offer the best explanation of the etiology of the disease. In more chronic cases the overgrowth of the neuroglia might be looked upon to some extent as supplementary (*vacatswucherung*), that is, the permanent vascularity being certainly not diminished and often increased, whereas the nerve elements have perished, there remains less tissue to nourish, hence the increased nourishment to the neuroglia leads to its overgrowth. Changes have been found in the neuroglia in quite recent cases, so that the irritation theory would appear best to account for the changes in the neuroglia, certainly in the earlier cases. In respect to etiology, it is interesting to note that micro-organisms have not been found in the cord. Yet the possible occurrence of epidemics of infantile paralysis as recorded by Medin (*Centrabl. f. klin. Med.*, 1891), and the experimental evidence as given by Roger (*Rev. de Méd.*, 1893), and more recently by Thoinet and Masselin (*Rev. de Méd.*, June, 1894), strongly support the view that the disease is an infective one. Thoinet and Masselin injected preparations of the B. coli communis and Staphylococcus, and determined spinal lesions at varying intervals after the inoculation, the peripheral nerves remaining intact. It is the poisons excreted by the micro-organisms that set up the disease, and this accounts for the early vascular phenomena found in the cord.

ON ENDOTHELIOMA OF THE SPINAL DURA MATER: WITH A CASE IN WHICH AN OPERATION WAS PERFORMED.

BY J. MICHELL CLARKE, M.A., M.D., M.R.C.P.

Physician and Pathologist to the Bristol General Hospital.

THE following case of endothelioma of the spinal dura mater presents several features of both clinical and pathological interest:—

A. P., aged 28, a bootmaker, came to the hospital on April 1, 1892, complaining of loss of power in the arms and legs. He had never had any previous illness. Eight years ago he had a sore on the penis, which was not followed by a rash, sore throat, or any other symptoms. In July, 1890, he had an attack of shivering, followed by fever, loss of appetite, and vomiting. There were looseness of the bowels and delirium, but after a few days he returned to work well. In July, 1891, he had a fall from his bicycle on to his left side, but apparently sustained no injury. When riding his bicycle in the early part of this year, he occasionally felt numb sensations in the legs. In September, 1891, the present illness began with twitching sensations in the hands on waking in the morning, passing off after breakfast. He also noticed a difficulty in holding small objects, and that when he had a tool in his hand he could not at once put it down. All these symptoms gradually grew worse, and the legs began to grow weak, until he had to give up work on December 3, 1891. Besides weakness of legs and arms, the muscles of the latter slowly wasted. With the exception of some dull aching pain in the shoulders, the loss of power was not accompanied by pain, and there were no shooting pains in either arms or legs. There appears to have been loss of sensation at this time, for he several times, whilst lighting the fire, burnt his left hand without being aware of it. He had had no

affection of sight or of speech, no attacks of sickness, no difficulty in breathing, micturition, or defæcation. The wasting of the arm muscles began later than the loss of power and sensory affections. He was positive as to the absence of severe pain throughout the illness and of injury to the spine.

Symptoms on Admission, April, 1892.—Patient was a tall, well-nourished man, of good general health. He spoke distinctly. There was no oculo-motor or facial paralysis. He walked badly, with an unsteady gait, dragging the left leg, which was kept stiff and rigid; he was unable to turn round sharply. He could not stand with his feet together and eyes closed. When lying down he could only lift the right foot with difficulty; the left hardly at all. He had great difficulty in picking up a pin with either hand, especially with the left, and he could not button his clothes. The fingers were flexed at the last two phalangeal joints. *Right hand:*—Movements feeble, especially in extensor indicis. *Left hand:*—Interossei and abductor indicis paralysed and somewhat wasted; slight atrophy of muscles of thenar eminence, with tendency of thumb to lie in ape-like position. The reaction of these muscles to faradism was feeble. He could bring his right hand up to his mouth, but not the left. The muscles of the left arm and forearm were smaller than those of the right. There was loss of sensibility over each forearm and outer part of each arm. The legs showed well-marked rigidity. The knee-jerks were exaggerated; ankle clonus present on each side; spontaneous clonic spasm of legs occurred at intervals on standing. The abdominal and cremasteric reflexes were at this time not obtained. No loss of sensation over lower extremities, nor over chest, but there was some deficient sensation over upper part of abdomen. Ten days after admission, whilst walking in the ward, he slipped and fell on his face; after the fall he became much worse, lost all power of walking or standing, and nearly all movement in his arms. Except for some aching and burning sensations in the limbs, which lasted only a short time, he had no pain after the fall.

May 1, 1892.—He could raise the shoulders and arms slightly, but could not depress the raised arm, nor lift the arms off the bed, and had absolutely no movement in the fingers, wrists, and forearms. There was complete loss of power in the legs, which lay in position of extension. The arms lay by his side, with the elbows flexed, the forearms across the epigastrium on their palmar aspect, the wrists flaccid, neither flexed nor extended, and the fingers flexed at the phalangeal joints. All the joints could be passively flexed and extended. The muscles of the back were decidedly weak.

There was marked clasp-knife rigidity of legs, with knee and ankle clonus. Plantar reflexes feeble—lower abdominal present, upper abdominal absent. No wrist-jerk; doubtful whether a feeble elbow-jerk was obtained.

Sensation was lost to all forms over both hands, except that a pinch could just be felt in the palms, and sensation to firm pressure was present over the backs of the second and third fingers of the right hand. Touch, pain, and temperature sense absent over backs of forearms; over palmar surface of forearms very deficient sensation to touch and pain; temperature sense preserved. The contact of a moderately hot test-tube with the back of the right arm raised a blister, which healed very slowly. Over the arms the temperature sense was absent; touch and pain were almost normally felt. Sensation was deficient on the chest from the second to the fifth ribs, and lost over an encircling band extending behind from the fifth to the eighth dorsal spines, from the spine of the scapula to one inch below its lower angle, and in front from the fifth to the level of the tenth rib. There was no loss of sensation over the head and neck, nor over the lower extremities. The bladder and rectum remained unaffected.

Three weeks later, a peculiar modification of sensory disturbance, resembling that found in syringomyelia, was observed over the arms and over the chest, from the clavicles to the level of the lower border of the pectoralis; on this area there was loss of sensation to pain and temperature, but complete preservation of the tactile sense, so that two points could be distinguished at the normal distance and the light contact of a single hair be detected. The sense of position was unaffected.

In July, 1892, the condition was much the same, with the exception that the wasting in the muscles of the upper extremity had certainly increased. Fibrillary tremor was present in the wasted muscles; those chiefly affected were the deltoids and small muscles of the hands, especially of the left hand. These muscles showed greatly diminished reaction to faradic and increased to galvanic currents. The other muscles of the shoulders, arms, and forearms were wasted, but to a less extent. He complained of burning and tingling pains under the finger-nails and at the finger-tips. Sensation same, with the peculiar disturbance over the limited area mentioned above. The diagnosis made at this time was that of pachymeningitis in the cervical region, with pressure on the cord, possibly of syphilitic origin. It was recognised, however, that the symptoms did not exactly correspond to those of pachymeningitis cervicalis hypertrophica,

especially in the absence of severe pain. The sensory modifications over the arms and chest suggested syringomyelia. He was treated with full doses of iodide of potassium, with mercurial inunction and counter-irritation over the cervical spine, but without any benefit, and after being in the hospital about ten months, as the disease seemed stationary, and no further treatment was at that time contemplated, he was discharged.

He returned to the hospital in July, 1894, with an urgent desire that something more should be done for him. Some friend had told him of the possibility of an operation affording him relief, and he wished that anything should be tried which would give him any chance of some recovery of power, if only the ability to feed himself.

Condition on Re-admission.—He was perfectly helpless. He lay on his back with his legs drawn up, and any stimulus such as withdrawing the bedclothes caused them to be still further drawn up. The right arm was slightly abducted, the elbow flexed, the forearm pronated and lying across the chest, the wrist was somewhat extended and the fingers flexed into the palm; the thumb was slightly flexed and adducted lying outside the fingers (modified "main en griffe"). The fingers and thumb could only be passively extended by using some force. Both extensor and flexor muscles were wasted; the latter the most so. The small muscles of the hand showed the greatest degree of wasting. The patient could accomplish very slight flexion, extension, and pronation of the forearm, and could raise the shoulder directly upwards to a small extent, but all these movements were very limited and of no practical service to him.

The left upper extremity was kept extended by his side, the forearm being prone. The fingers and wrist lay in the same position as on the right side; he had no power of movement in this limb. The legs were rigid, the muscles being hard, and not wasted; the patient could slightly flex them at the hip and knee-joints, but had no power over the ankles or toes. The leg muscles reacted to both faradic and galvanic currents. The head and neck could be moved freely in any direction. There was no paralysis of any cranial nerve. He had no control over the bladder, and suffered from obstinate constipation. The finger-nails showed trophic changes, being white and brittle.

Reflexes.—Abdominal, plantar and cremasteric all increased. Knee-jerks exaggerated, especially on the right side; patellar and ankle-clonus easily obtained.

Electrical Reactions.—Faradic current: right arm. No reaction in deltoid; long flexors react very feebly; no reaction in

small muscles of hand; other muscles react well; biceps reacted better than triceps. Left arm.—No reaction obtained for pectoralis major, deltoid, biceps and intrinsic muscles of hand; other muscles contract.

RIGHT ARM.

Constant Current.

Biceps	.. KCC = 3 milliampères..	ACC = 5 ma.
Pectoralis ..	.. KCC = 20 ma.	
Deltoid	.. KCC = 10 ma.	.. ACC = 9 ma.
Triceps.. ..	.. None with Kathode	.. ACC = 15 ma.
Long extensors of fingers..	KCC = 2 ma...	.. ACC = 5 ma.
„ flexors „	.. No reaction ..	.. ACC = 12 ma.
Dorsal interossei	.. KCC = 2·5 ma.	.. ACC = 5 ma.
Thumb muscles ..	.. No reaction.	

LEFT ARM.

Constant Current.

Biceps	.. No reaction up to 20 ma. of current.	
Pectoralis ..	.. KCC = 10 ma.	.. ACC = 0.
Deltoid	.. No reaction.	
Long extensors of fingers ..	KCC = 4 ma...	.. ACC = 4·5 ma.
„ flexors „	.. KCC = 15 ma.	.. ACC = 15 ma.
Dorsal interossei	.. KCC = 12 ma.	.. ACC = 12 ma.
Thumb muscles ..	.. No reaction.	

From the accompanying charts the disturbances of sensation present at this period of the case can be seen. The peculiar retention of tactile with loss of pain and temperature sense over a certain area observed earlier in the case was now absent. On this account several careful investigations were made of the state of sensation to heat and cold, the results of which are given in figs. C and D, which show curious anomalies of the temperature sense over the body surface. The diagnosis at this time seemed on the whole to be in favour of a swelling external to the cord and pressing upon it. As there was no sign of disease of vertebræ, this was not likely to be a mass of caseous tubercular material, such as is found compressing the cord in tubercular caries. The absence of any improvement under treatment by iodide of potassium, and of positive evidence of syphilis, rendered a gumma or syphilitic inflammatory swelling of the membranes improbable. The duration of the illness, and the fact that during the last eighteen months the paralysis of sensation had remained stationary, whilst in the case of the motor paralysis there had been slight improvement in the arms, negatived the existence of tumour of a malignant character. It was considered that the sudden increase of paralysis after a fall during his previous stay

in the hospital was due to a patch of myelitis at the seat of lesion.

Both motor and sensory symptoms indicated that the lesion lay from the origin of the fifth to the eighth cervical nerve roots. The symptoms pointed to considerable vertical extent of the lesion of the cord, and on this account the probability of a growth seated centrally within the cord had to be taken into consideration,

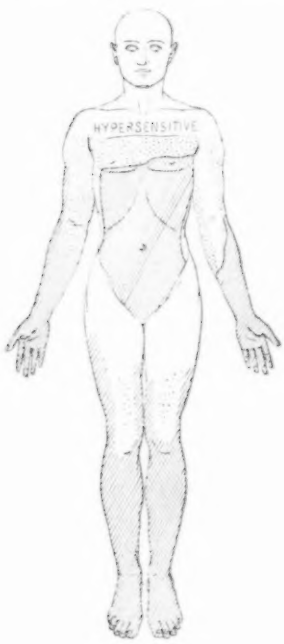

FIG. A.

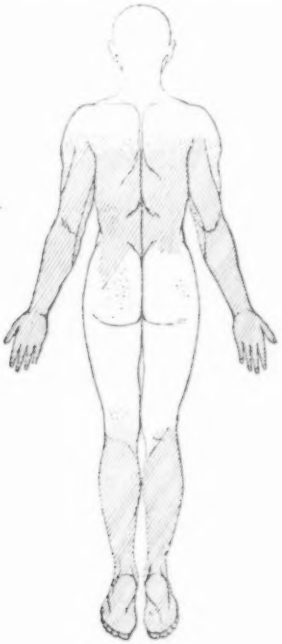

FIG. B.

but the long duration and stationary character of symptoms precluded a tumour of this kind, which is generally a glioma. Considerable vertical extent of lesion is also found in cases of meningitis, and the cervical region of the cord is the common seat of hypertrophic pachymeningitis, with the symptoms of which those of my patient corresponded fairly well, but for the important exception of absence of the severe radiating pains generally present in this disease.

An exact diagnosis of the nature of the disease was thus unfortunately not arrived at, but the condition was considered to be a benign tumour of uncertain nature, or a localised thickening, probably seated in or outside the spinal membranes, and compressing the cord. Dr. J. A. Ormerod, who was in Bristol for the July meeting of the British Medical Association, 1894, kindly saw the case with me, and concurred in this view. The patient, who

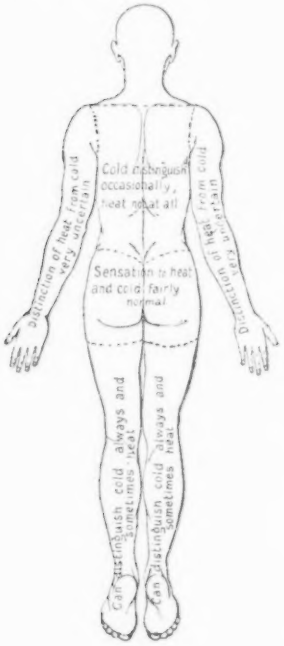
Distinction of heat from cold very uncertain
Cold distinguish occasionally, heat not at all
Distinction of heat from cold uncertain
Sensation to heat and cold fairly normal
Can distinguish cold always and heat sometimes
Can distinguish cold always and heat sometimes

FIG. C.

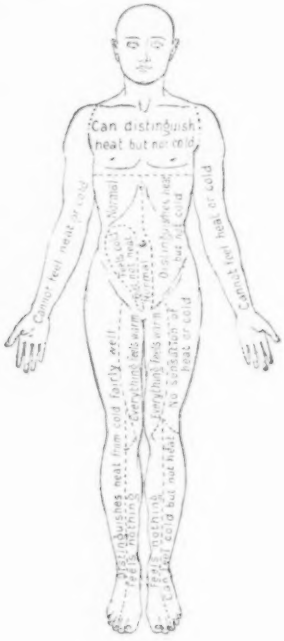
Can distinguish heat but not cold
Cannot feel heat or cold
Cannot feel heat or cold
Normal
Everything feels warm
Everything feels warm
Can distinguish heat but not cold
Can distinguish heat but not cold
Can distinguish heat from cold fairly well
Can distinguish heat but not cold
Can distinguish heat but not cold
Can distinguish heat but not cold
No sensation of heat or cold

FIG. D.

was a very intelligent man, was very anxious that something should be done for him, and the dangers of an operation and the possibility that no good might result from it were put plainly before him. I decided to wait a month, at any rate, until I returned from my holiday, and that he should fully consider the question in the meantime. The fact that he had regained some slight power in the right arm and that most muscles in the arms still retained some electrical irritability, led me to hope that if

the pressure could be removed, some recovery in the arms might ensue, even after the long duration of symptoms, and that the spasmodic contractions of the leg muscles, which gave him much pain, would be relieved. On my return, he still strongly wished that an operation should be attempted, and my colleague, Mr. W. M. Barclay, accordingly operated on September 12. He has kindly furnished me with the following notes:—

"On September 12 I explored the cervical region of the cord. The patient was anæsthetised with chloroform, and placed in the semiprone position, and an incision was made over the spines from the fourth or fifth cervical to the first dorsal—this being subsequently extended a little downwards. The muscles were rapidly detached from the spines and laminæ, first on one side and then on the other, by the knife—the vertebral aponeurosis being transversely divided on each side to allow of freer retraction of the muscles, as recommended by Mr. Horsley. The bleeding was very excessive and very difficult to stop, and the patient lost a great deal of blood before this was accomplished. The sixth and seventh cervical spines were then cut off by bone forceps, and the seventh laminal arch trephined—the arches of the sixth, fifth, and fourth being subsequently removed by forceps. On thus exposing the spinal canal, it was found to be completely filled with a reddish-grey, smooth material looking like the reddened dura mater. When, however, I incised the supposed dura mater, I cut into a greyish granular and gelatinous-looking substance, which proved to be entirely outside the dura; it extended as far up as the fourth lamina, possibly further, and dipped under the first dorsal arch below; on each side it passed forwards round the cord among the nerve roots, and could not be further exposed. The patient had been getting very faint for some time, and at this stage he became so collapsed that I was obliged hastily to cut away a small part of the mass on the dorsal aspect and close the wound. With warmth and injections of brandy, hypodermically and *per rectum*, he seemed to rally somewhat, but the collapse returned, and he sank the same night.

"The excessive loss of blood at the early stage of the operation was most untoward, and unlike any I had previously seen in clearing the spine. It seemed unavoidable, and I cannot see what measures could have been adopted with a better chance of success. A still further delay was unfortunately caused by the forceps (Victor Horsley's laminectomy forceps) being unable to bite through the laminæ. In consequence of this I had to use ordinary bone forceps, which are very clumsy for this work.

"I had hoped (had he survived the operation) that by removing the dorsal part of the mass around his cord, the pressure might be, to some extent, relieved, and the patient's condition ameliorated."

The autopsy was made seventeen hours after death. There was the scar of an old bed-sore over the sacrum. The operation wound in back of the neck looked healthy. The posterior arches of the fourth, fifth, sixth, and seventh cervical vertebræ had been cut away, and the dura mater lay exposed in the floor of the wound, in which lay a little blood. On laying open the spinal canal, dura mater and cord appeared quite normal under the wound, except for a minute fragment of pale growth immediately below it. Above the wound the cord was covered posteriorly by a flattened layer of somewhat friable new growth, which extended around it forwards so as almost to enclose it; the growth lay outside the dura mater, and was adherent to the inner surface of the posterior arches of the third and fourth cervical vertebræ. The membranes were adherent to each other and to the cord over the entire extent of the tumour, and the cord was also very soft for a corresponding distance, and was decidedly flattened from before backwards. Owing to its softness and to the surrounding adhesions, it was difficult to remove, and was slightly torn. The growth did not extend on to anterior surface of cord at level of operation wound, but did so slightly above it. The cord, which, to the naked eye, in the fresh state, showed no degenerated tracts, was at once placed in Müller's fluid. The vertebræ were everywhere normal in all respects; there was no sign of caries, of old fracture, or growth in them, and the tumour was not connected with them.

On further examination after hardening, the tumour was found to extend from the origin of the fifth cervical nerve roots to that of the first dorsal. It lay chiefly in the posterior aspect of the cord, which it had flattened and compressed throughout nearly the whole of the cervical enlargement. Its greatest bulk was on the left side of the posterior aspect of the cord; the nerve roots were somewhat compressed and atrophied from pressure by the tumour, perhaps especially on the left side; but all those corresponding to the vertical extent of the tumour were more or less affected. It lay external to the dura mater, appeared to be loosely attached to it, leaving a rough surface where removed. There was nowhere any trace of hæmorrhage into the tumour, which was soft, and not tough, nor was it laminated in structure. It was about 2¼ inches long, of which a piece, about 1 inch in length, was removed at the operation.

Its breadth on removal, extending round cord, was $1\frac{1}{4}$ inches; its average thickness $\frac{1}{4}$ inch, greatest thickness, $\frac{3}{8}$ inch. The membranes under the growth were adherent to each other to the cord.

Circumference of spinal cord $\frac{3}{4}$ inch above upper edge of growth	$2\frac{1}{4}$ inches
" " " " at middle of growth about middle of cervical enlargement	$1\frac{1}{2}$ inches
Circumference of spinal cord, end of cervical enlargement	$1\frac{7}{8}$ inches

These measurements show the extent of the compression of the cord.

Sections of the cord were stained by Weigert's method, and with carmine and logwood.

Throughout a vertical extent in correspondence with that of the growth, the cord had undergone extensive destruction from pressure, and at the point of greatest softening its different structures were distinguished with difficulty; but except for a very short distance the main features of its structure were clearly recognisable. It was noticed that although both grey and white matter had undergone extensive destruction, in sections stained by Weigert's method there were found many fibres in the white columns which had retained their myelin sheaths. These fibres were fairly numerous in both the posterior and anterior, and lateral columns, but especially in the latter and were present in all sections, including those from the part which had suffered greatest compression.

Weigert's stain also brought into view ascending degenerations, chiefly in the posterior columns, above the tumour, and descending degeneration in the pyramidal tracts below it, although these were not visible in the fresh cord. Neither in the ascending and descending tracts was the degeneration complete, in all of them about a third at a rough estimate of the total number of fibres still retained their myelin sheaths and were to all appearance healthy. There was no growth within the membranes or within the spinal cord itself. In the upper part of the cervical region one or two longitudinal slits were present in the posterior part of the cord near the base of the posterior horn; but careful examination showed that these were produced during the removal of the cord.

Portions of the tumour were embedded in celloidin and sections stained, some in logwood and resin, and others in picro-carmine.

Microscopically the tumour was seen to spring from the outer surface of the dura mater; its structure is represented in the figures. It belonged to the endotheliomata or psammomata, more correctly speaking to the former, as there were no calcareous granules in it. The stroma was composed of rounded or oval nucleated cells lying in a partly structureless, partly indistinctly fibrillated matrix. Passing through the tumour were vessels and also trabeculæ. In some parts the cells had a roughly alveolar or lobular arrangement, apparently determined by the course of the vessels. Scattered through the tumour were endothelial cell nests. The individual cells composing these could not be made out, save in one or two places where they were in process of formation. These nests seemed to be of slow growth, if one may judge from a comparison of their structure with the richly nucleated endothelial cell nests in a case of psammoma of the cerebral dura mater under my care (see figs. 4 and 5). Outside the endothelium of the small vessels was a layer of homogeneous hyaline-looking material, in the position of the muscular coat, but thicker than a normal muscular coat, and also present in the smallest vessels. This stained pink with picro-carmine, as did also some bands of similar appearance which passed through the tumour and did not contain vessels. The cell nests show the same hyaline material in their middle and external layers. Ernst, in a paper on psammoma ("Ueber Psammoma," *Beiträg. z. Path. Anat. und z. Allgemein. Path.*, 1891-2, p. 234), describes and figures the band of hyaline material which forms in place of muscle a hyaline layer outside the endothelium, and also thicker bands of the same substance running through the tumour. He regards the hyaline substance as a change antecedent to calcification. This paper gives an excellent account of the best methods of staining and of various questions as to the nature and genesis of psammoma.

This hyaline change in vessels and cylinder-like bodies is also described by Drs. Bremer and Carson ("Cylindroma

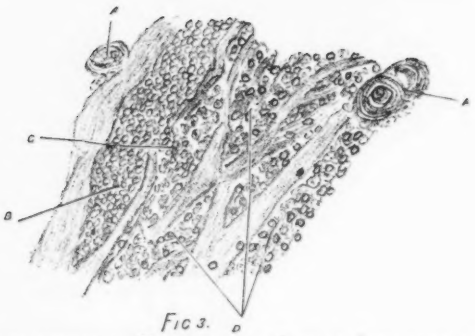

FIG 3.

SHOWS SPACES FORMED BY TRABECULÆ OF SIMILAR STRUCTURE TO VESSEL WALLS CONTAINING CELLS OF STROMA AND NUMEROUS RED BLOOD CORPUSCLES.

A A—Cell nests. B—Vessel with narrow imperfect wall on one side through which blood corpuscles are escaping at C. D—Spaces containing numerous blood cells.—*Zeiss*, Apochrom. 4 mm. Compens. Oc. 4.

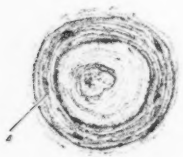

FIG 4.

CELL NEST MORE HIGHLY MAGNIFIED.

A—Layer resembling in appearance wall of vessel.

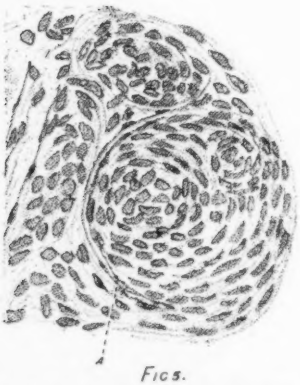

FIG 5.

CELL NEST FROM CASE OF PSAMMOMA OF CEREBRAL DURA MATER.

Comparison of figures shows richness of latter in nuclei. Cell nests in former smaller, and neither with logwood nor picro-carmine were nuclei brought out; with logwood nests appeared homogeneous in outer layers, staining a very intense blue. Cell nest in fig. 4 is thus of slower growth than that in fig. 5.—*Zeiss*, Apochrom. 4 mm. Compens. Oc. 8.

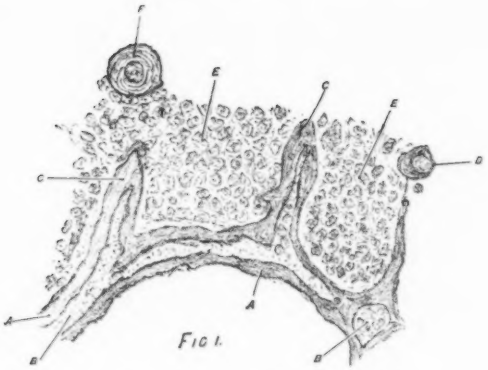

FIG 1.

VESSEL WITH HYALINE HOMOGENEOUS WALLS.

A A—Vessel wall. B B—Lumen with blood corpuscles. C C—Buds given off from vessel. D—Small cell nest of similar hyaline substance apparently attached to vessel by slender thread. E E—Cells of stroma. F—Cell nest.—*Zeiss*, Apochrom. 4 mm. Compens. Oc. 4.

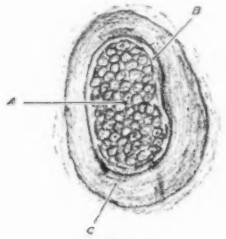

FIG 2.

VESSEL MORE HIGHLY MAGNIFIED.—*Zeiss*, Apoch. 4 mm. Compens. Oc. 8.

A—Lumen of vessel. B—Endothelial layer (? broader than normal). C—Hyaline coat of vessel.

Endothelioides of Dura Mater," *Amer. J. Med. Sci.*, Feb., 1895, p. 120), and they also figure as "capillary angioma" in fig. 4 of their paper much the same appearances that I have drawn in fig. 3. The walls of these blood spaces appear to consist of pink stained homogeneous hyaline substance. As to staining I may briefly mention that picro-carmine gave very good results for the vessels and cell nests; logwood stained the cells of the stroma well, but the cell nests were inconspicuous in logwood stained sections, and their true nature could only imperfectly be distinguished. I made my sections before reading Ernst's paper, and could not follow the method there recommended and quoted from Van Gieson (*N.Y. Med. Journ.*, July 20, 1889) of staining with a mixture of acid fuchsin and picric acid and for nuclei of cells with Dalafield's hæmatoxylin. Picro-carmine gave, however, very good results indeed, especially when supplemented for cells of tumour-stroma by a comparison of sections stained in logwood.

Endothelioma, or psammoma, seems to be relatively of more seldom occurrence in the spinal than in the cerebral meninges. No doubt some of the cases described in the older writings on tumours of the spinal meninges as "fungus of the dura mater," epithelioma and fibroma of the dura mater include examples of the different forms of psammoma or endothelioma. Wilks and Moxon mention ("Pathological Anatomy," p. 249), without further particulars, having met with one case of psammoma of the spinal membranes, and I have found the following cases in a fairly extensive search through the literature. It seemed, however, useless to go very far back, as the microscopic structure of tumours of older date was not clearly differentiated.

(1) J. Hutchinson, Junr. (*Path. Trans.*, vol. xxxiii., p. 23): Woman, aged 48, tumour $1\frac{1}{4}$ in. by $\frac{1}{2}$ in. in dorsal region of cord; microscopic calcification had occurred in it. Duration of symptoms two years; paraplegia without loss of sensation; very severe and constant pains shooting into lower part of abdomen and down legs.

(2) Mr. J. Hutchinson refers to a case described by Dr. Wilks (*Path. Trans.*, vol. vii., p. 37) as a tumour of fibro-

nucleated structure, not simple fibrous growth, in a woman aged 43. Tumour size of a bean, of oval shape, firm, hard, and grey, and growing from dura-arachnoid. Symptoms of nine months' duration, began with pains between shoulders and in chest and legs. She suffered from weakness of legs, but without complete paralysis and without sensory affection, and from violent pains and convulsive twitchings in limbs and abdomen. Dr. Wilks adds to the account of this case that: "In the year 1849 a man died in Guy's Hospital from a very similar tumour of exact form and size, and growing from the like part of the cord."

(3) H. A. Lediard (*Ibid.*, vol. xxxiii., p. 27): Tumour of dura mater pressing upon cord in cervical region. A note added to description says that specimen is in museum Royal College of Surgeons described as endothelioma connected with pia mater of spinal cord. Man, aged 25: duration of symptoms seventeen months. Pain and stiffness in neck; numbness, loss of power in left upper extremity; right arm next involved in same way, until finally there was complete paralysis of arms, legs and trunk. Death from implication of muscles of respiration. Tumour smooth, firm, pinky-white, irregularly lobed. Length $\frac{7}{8}$in. by $\frac{5}{8}$in. broad by $\frac{3}{8}$in. thick.

(4) Dr. William Cayley (*Ibid.*, vol. xvi.): Oval tumour at interval between tenth and eleventh dorsal vertebræ growing from inner surface of dura mater, not adherent to cord; tumour soft, and white in colour. Woman, aged 46: duration of symptoms twelve months; $1\frac{1}{4}$in. long, by 5 lines thick. Calcified cells in centre of nests.

(5) T. Whipham (*Ibid.*, vol. xxiv., p. 15): Tumour opposite sixth and seventh cervical vertebræ, grew from dura mater, and was firmly adherent to it; though lying in such close proximity with cord, had no connection with it. The patient was a woman, aged 34: duration of symptoms, which began in legs, thirteen months. There is also a case reported by Dr. Ogle as carcinomatous tumour of foramen magnum (*Trans. Path. Soc.*, 1852-53), but no account of the microscopic structure of the growth is given.

Although the number of recorded cases of psammoma,

or endothelioma of the cerebral meninges is very large, the above are all the cases of growths in the spinal membranes that were certainly of this nature that I have been able to find in the literature accessible to me. I have, however, been unable to peruse two or three papers, especially one referred to by Ernst ("Ueber die primitiv geschwülste der Dura Mater," *Wein. Med. Jahrbucher*, 1874), which is based on twenty-eight cases, and one by MacAlister, "Sarcoma Spin. Meninges," Zurich, 1890. These papers may possibly contain more examples.

It will be noticed that these cases occurred in adults, and that the duration varied from nine months to two years. In none of them did metastatic growths occur, the tumours remaining localised, and not growing to any great size. Such cases would presumably be well suited for removal by operation. It would be interesting in future cases to have more information as to the previous occurrence or not of syphilitic infection and of blows or falls upon the back.

To recapitulate the most important features in my case; these were:—

(1) The long duration, with sudden exacerbation of all the symptoms after a fall.

(2) The close resemblance of the sensory disturbance over a certain area to that commonly met with in, and regarded as typical of, syringomyelia. This at the time caused some difficulty in diagnosis.

(3) The remarkable absence of severe pain, and of root-pains, radiating from the affected part of the cord; possibly accounted for by the soft nature and slow progress of the growth producing only very gradual increase of pressure on the nerve roots. This may possibly be in future an important diagnostic point in differentiating this variety of tumour from other growths in the spinal membranes.

(4) The existence after such a considerable time of many apparently healthy fibres in the affected part of the cord and in the degenerated tracts, presumably rendering possible a fair amount of recovery. The operation, however, did not remove the whole of the growth.

A CASE OF CEREBRAL TUMOUR, WITH HEMI-ANÆSTHESIA.

BY H. J. MACKAY, M.D.

JAMES M., aged 45, a labourer, was admitted into Devizes Cottage Hospital on November 20, 1893, complaining of severe headache, inability to walk, and numbness, with some weakness of the left side. His family history was unimportant; his personal record showed that he had chancre in his twentieth year, and malarial fever when on foreign service; otherwise his health had been good, and he had not drunk to excess. His present illness began nine weeks before, with severe pain in the head; he had, however, noticed giddiness and occasional headaches for some months before. Two weeks after the severe headache began he had cramp in his legs, chiefly in the calves, but they did not jerk, merely becoming stiff and painful. During an attack of this sort, seven weeks before admission, he fell downstairs, striking his right ear and the head behind the ear in the fall. A month later he had a shivering attack with intense aching in the back, neck and head (? influenza), for which he went to bed, and remained there till his admission to the hospital. There were no convulsions or motor spasms at any time during his illness, nor did he ever lose consciousness. While in bed he noticed a gradual onset of numbness in the left side of his body, which also became somewhat weaker than the right; coincident with this his wife had noticed increasing torpor and stupidity. He had had severe attacks of vomiting, independently of taking food. He complained also of giddiness, and of failure of sight.

Examination showed the patient to be a well-built, muscular man, who lay on his back with closed eyes, seemed stupid and heavy, and appeared to have difficulty in understanding and answering the questions put to him. When pressed to answer he would, after a pause, say impatiently, "He did not know," or make a quite irrelevant reply. The information he volunteered, however, apart from questioning, was intelligent, coherent, and to the point. He never mixed up incongruous words in his sentences, nor made meaningless sounds. He could read the letters in Snellen's types, and write small words correctly. His condition therefore suggested that of sensory aphasia, of the variety described as word-deafness. He complained greatly of pain in the frontal region, and cranial percussion elicited tenderness over the right temporo-parietal region. His motor power was good on the right side but slightly impaired on the left. He had still, however, considerable power in the left hand grasp, and in flexing and extending the left leg. Muscular nutrition was everywhere good. There was no rigidity on passive movement. Electrical irritability was diminished to both currents on the left side of the body, but there was no qualitative change.

The knee-jerks were normal on both sides, and there was no clonus. The plantar and cremasteric reflexes were impaired on the left side. Bladder and bowel control were normal. The right pupil was dilated to about No. 12 catheter gauge, and did not react to light; the left was of medium size, and re-acted but sluggishly. Co-ordination was unaffected on the right side, but appeared defective on the left.

Muscular sense is much impaired on the left side. He is quite unaware in what position his left arm and leg are placed, imagining them to be straight when they are flexed: this is not the case with the right side.

There is great impairment of sensation to touch, pain and temperature over the left side of the body. He does not feel a pin-prick, which on the right side makes him wince and withdraw the limb. He does not recognise as warm a test tube of water a little below boiling point. He is quite

unconscious of light touches over the impaired area. This includes the left half of the face and trunk and the left arm and leg, and the anæsthesia appears to be most marked in the leg. Normal sensation is recovered at about an inch to the right of the mesial line, and continues over the right side of the body.

The condition of the cranial nerves is as follows:—

I. He cannot distinguish in either nostril, tested separately, the smell of hyacinth, cloves, peppermint or tobacco. He complains that there is no flavour in his meat or his tea, and that everything tastes alike.

II. Vision, R. $\frac{6}{65}$. J.o. at 22 cm.

L. P.L. only.

There is optic neuritis on the right side. On the left the disc cannot be examined on account of cloudiness of the cornea. There is concentric contraction of the right field. The right pupil is dilated and motionless, the left is of medium size and probably reacts sluggishly to light. Reaction to accommodation cannot be made out in either eye.

III., IV., VI. } Ptosis and paralysis of internal rectus on right side.

V. Nearly complete facial anæsthesia of left side, which includes the mucous surfaces and extends slightly to the right of the middle line. Commencing neuroparalytic ophthalmia in left eye. Masseteric contraction equal on both sides.

VII. Unaffected.

VIII. He hears a watch tick which is normally audible at thirty inches, at a distance of two inches in the right and twelve inches in the left ear. The relatives cannot say whether he was deaf before his illnes, but think he was not. The tympana are healthy in both ears.

IX. He cannot distinguish on either side of the tongue solutions of salt, tartaric acid, quinine or sugar. The tongue is thickly furred. The solutions are applied to the back as well as the front of the tongue.

X., XI., XII. } Unaffected.

There is some swelling of the left side of the face, and a bright flush on the left cheek.

T. 99°. P. 64, full, laboured, regular. R. 18.

The urine is acid, specific gravity 1,018, free from albumen and sugar.

On the 23rd it was noted that the ptosis was less marked, and on the 24th the right eye was opened equally with the left, and the right internal rectus had regained its power. The right pupil was still dilated, but now reacted sluggishly to light.

During his stay in hospital the observations made were frequently verified. He was frequently seen to move all his limbs, and to raise himself and sit on the side of the bed. He was unable to stand or walk, on account of vertigo. Subsequently the ophthalmia of the left eye went on to perforation of the cornea, and his torpor deepened; he had still, however, occasional intervals of clearness, when he would ask questions quite intelligently, but could hardly ever be got to return a relevant answer. On December 12 he became comatose, his temperature rose to 106·2°, and he died shortly afterwards.

Autopsy fourteen hours after death, at which I was assisted by Dr. Adie. On opening the skull there was thickening and opacity of the dura over the right temporo-sphenoidal lobe. The arachnoid space contained several ounces of blood-stained serum, which was seen to ooze from the sulcus surrounding a tumour in the right lower temporo-sphenoidal region, and the freedom of the flow suggested communication with the right lateral ventricle.

The tumour (fig. 6) was lenticular in shape, measuring 4·5 cm. in diameter each way, by 2 cm. in thickness. It occupied a bed of softened brain tissue which had absorbed half the breadth of the middle, and had completely obliterated the lower temporo-sphenoidal convolutions. The superior T. S. convolution was greatly enlarged in breadth and length, and was, like the remainder of the brain, displaced upwards and forwards. Owing to this displacement the frontal and parietal lobes were compressed somewhat, and the fissure of Sylvius was anterior to its

usual position. Moreover, the T. S. lobe occupied more than its normal proportion of the surface-area of the hemisphere. The left hemisphere was natural in shape and proportion. The nerves at the base of the brain showed no change beyond compression and flattening of the right oculo-motor. No naked-eye change could be detected in the fifth of the left side, nor in the Gasserian ganglion, nor in the auditory of the right side. Unfortunately these structures were not preserved for microscopical examination, as was intended.

On the median aspect of the right hemisphere there is evident compression and softening of the hippocampal gyrus in the neighbourhood of the isthmus, and between this and the uncus. The uncinate, and occipito-temporal gyrus (beneath the collateral fissure), are also superficially softened. The lingual gyrus appears, outwardly, unaffected. The rest of the median surface of the hemisphere appears normal. A vertical (frontal) section was made through the hemisphere in the plane of the centre of the tumour (line A B in fig. 6). This plane passed through the splenium of the corpus callosum in its greatest diameter, and met, at the superior margin of the hemisphere, the posterior and superior extremity of the fissure of Rolando.

It was seen by the section (fig. 7) that the occipito-temporal gyrus was compressed to a mere shred, from 2 to 3 mm. in its greatest thickness, by the tumour. The medullary matter of the lingual gyrus, and that at the bottom of the calcarine fissure (which includes the medullary matter of the hippocampal gyrus at this spot), was softened to pulp. The bed of the tumour occupies most of the thickness of the temporo-sphenoidal lobe in its lower two-thirds and its posterior half; and from the softened medullary matter composing it a strand of softened tissue passes upwards and inwards towards the floor of the lateral ventricle. This softened tract extends up both walls of the lateral ventricle; on the outer side as far as the grey matter forming the bottom of the parallel fissure; and on the inner side, formed here by the cornu ammonis below, and higher up by the alveus of the hip-

pocampus (posterior pillar of the fornix), the softening extends as far as the splenium of the corpus callosum, which is not softened. A small fragment of the posterior extremity of the optic thalamus, which the section includes, lying subjacent to the splenium, is also unsoftened. The cavity of the ventricle is obliterated, and the softened walls are in contact with each other.

A horizontal transverse section through the anterior portion of the hemisphere, in the plane of the line E F (fig. 9), displayed the internal capsule in relation to the softened medullary matter of the T. S. lobe. It was found that the posterior fibres of the capsule did not share in the softening, but were separated from the disorganised tissue by a space, a few mm. in width, of apparently healthy medullary matter. Beyond this distance (about five mm. from the posterior extremity of the lenticular nucleus), the medullary fibres were softened and were in anatomical continuity with the softened medullary substance forming the bed of the tumour. A softened tract of arcuate fibres could also be traced passing into the white matter of the adjacent (superior) T. S. convolution.

Sections in, and anterior to, the plane of C D did not reveal any fresh facts, and sections in the plane of A B through the hinder portion of the brain showed that the area of softening did not extend posteriorly beyond the region of actual compression by the tumour.

The fibres of the crusta and tegmentum of the cerebral peduncles showed no visible change, nor did the longitudinal fibres of the pons.

The brain was hardened in Müller's fluid for two months, and for a month in alcohol. Sections were then cut from: (1) the tumour; (2) the posterior fasciculi of the internal capsule; (3) the tract of medullary substance posterior to this, endeavouring to include the boundary line between healthy and softened tissue; (4) the softened hippocampus; (5) the superior T. S. convolution in its middle undamaged portion; (6) the right crus-cerebri and pons.

(1) The tumour was found to be enclosed, on its outer

surface, in a capsule of vascular connective tissue, which appeared to be thickened pia. Internal to this was a zone containing numerous arterioles surrounded by connective tissue which was densely infiltrated by small-celled leucocyte-looking bodies which took on a deep stain, and resembled the granulation tissue of syphilitic growths. The arterioles in this zone and in the capsule were for the most part either obliterated or in process of being obliterated, by the invasion of the outer and inner tunics by the small-celled growth mentioned. Many of those so obliterated showed the presence of white fibrous tissue more or less replacing the walls of the vessel, and some of them on section had the appearance of being round cords of fibrous tissue. More internal to this granulation zone were areas of loose connective tissue, and strands of dense fibrous tissue, the latter sometimes enclosing tracts of hæmorrhagic *débris*, and elsewhere of caseation.

(2) The posterior fibres of the internal capsule showed no morbid change. The nerve fibres were of equal size; their axis cylinders took on a deep stain with aniline blue black. The unstained white sheaths exhibited no enlargements nor degeneration.

(3) The tract of softened tissue posterior to the internal capsule could not be included in sections even when cut in celloidin. The broken edges from which tissue had disappeared exhibited at the boundary line swollen and irregular fibres, and numerous dark stained connective tissue nuclei. Sections teased in osmic acid showed abundant myelin globules in the irregular fibres, compound granule bodies and fatty *débris*.

(4) The hippocampal sections showed a total disappearance of the longitudinal fibres forming the alveus, and an absence of the large pyramidal cells from the subjacent layer. There appeared to be a great increase of connective tissue, with darkly stained nuclei, but the normal arrangement of the other layers in this part of the brain could not be recognised.

(5) The superior temporo-sphenoidal convolution was not softened anywhere except in the medullary (arcuate)

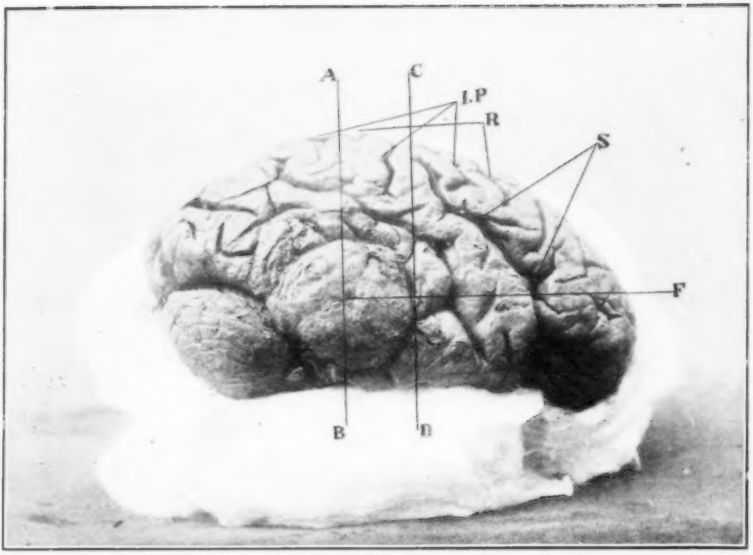

Fig. 6.

Right Hemisphere, with Tumour *in situ*. From photograph.

To show the tumour more clearly, the point of view is from a little below the transverse axis of the brain. Consequently the upper margin, including the termination of the fissure of Rolando, is not visible. I.P points to inter-parietal fissure; R and S, fissures of Rolando, and of Sylvius; the lines refer to planes of section mentioned in the text.

Case of J. M.

Section through hemisphere in plane of line AB. From a photograph, nat. size.

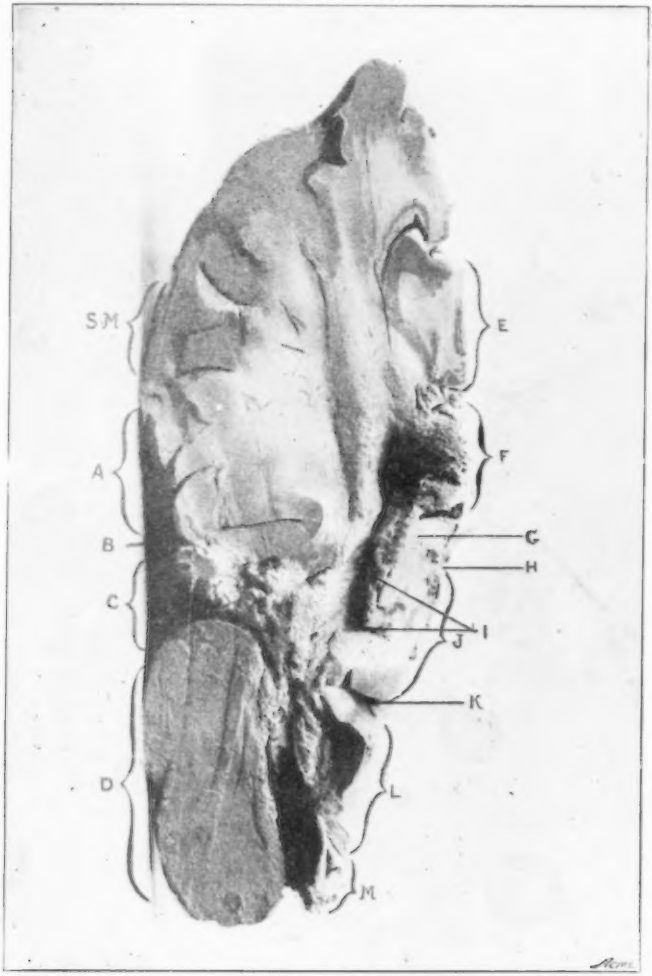

Fig. 7.

(The letters refer to figs. 7 and 8.)

SM—Supra-marginal Convolution; A—Angular Convolution; B—Parallel Fissure; C—Middle Temporo-sphenoidal Convolution; C¹ (in fig. 8)—Inferior T.S. Convolution; D (in fig. 7)—Tumour; M—Occipito-temporal Gyrus; L—Lingual Gyrus; K—Calcarine Fissure; J—Hippocampal Gyrus; I—Hippocampus Major; H—Hippocampal Fissure; G (in fig. 8)—Posterior Extremity of Optic Thalamus.

Section in same plane as in fig. 7, for comparison. From a photograph, nat. size.

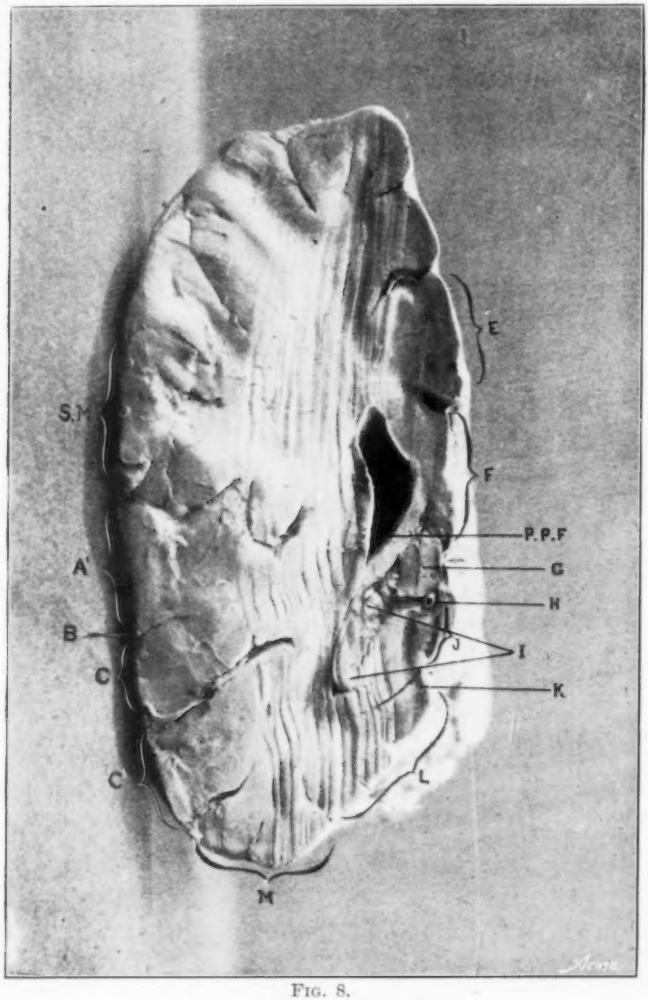

Fig. 8.

(The letters refer to figs. 7 and 8.)

P.P.F—Posterior Pillar of Fornix, descending to become the Alveus of the Hippocampus. The same structure, but disorganised, can be traced in fig. 7; F—Splenium of Corpus Callosum. The softened appearance in fig. 7 is due to compression by cotton wool, which was packed into the upper part of the Ventricle; E—Callosal Convolution.

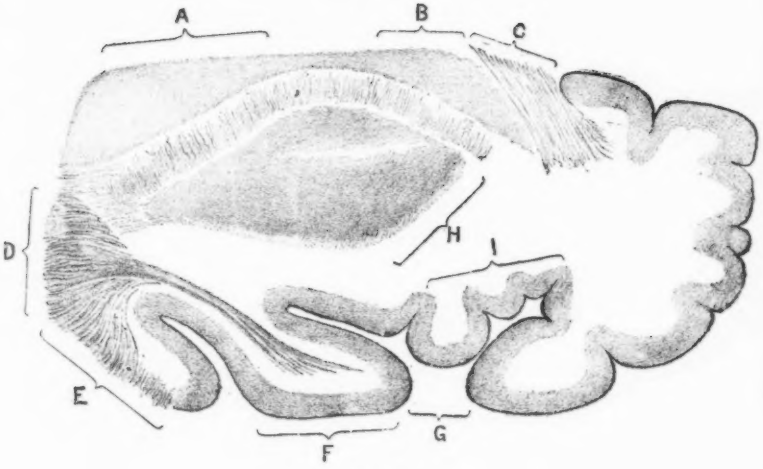

Fig. 9.

Horizontal transverse section through anterior portion of Hemisphere in plane of line E F. (From a tracing.) The softened medullary tissue at D is continuous with E, the softened bed of the tumour. There is seen to be an interval of unsoftened medullary substance posterior to the internal capsule. H– Lenticular Nucleus; B– Caudate Nucleus; A—Optic Thalamus; C –Commissural fibres of genu of corpus callosum; F– 1st T.S. Convolution, into which passes a strand of softened fibres; G— Fissure of Sylvius; I—Insula.

fibres in its anterior portion already referred to. Elsewhere there was a great increase in the size, and in the relative mass, of the neuroglial fibres and cells. The nuclei were more numerous than in normal sections and were occasionally double.

(6) The crus cerebri and pons exhibited normal fibres, with unaltered sheaths and axis cylinders on transverse section.

The sections were stained for the most part by logwood and carmine, by Bevan Lewis's method, and by osmic acid. I regret that my want of skill as a microscopic draughtsman does not allow me to reproduce their appearance.

Remarks.—The dearth of clinico-pathological records, throwing any light upon the localisation of the cerebral sensory areas, has been frequently remarked. The reproach cannot even yet be said to be in a fair way of being removed—a search of the only available literature at my disposal having yielded but scanty notes of cerebral cases in which tactile anæsthesia was associated with cortical lesions.

Ferrier[1] reports a case in which a tumour pressing on the left temporal and parietal lobes caused right hemi-anæsthesia. Vialet[2] mentions two cases of left hemianopsia in one of which right hemi-anæsthesia was present. In both of these there was atrophy of the convolutions bounding the calcarine fissure, atrophy of "the central portion of the parieto-occipital fissure," and atrophy of the foot of the cuneus extending to the lower extremity of the hippocampus; unfortunately no detailed criticism is given of the association of symptoms with lesions in each individual case.

Dana,[3] in a collection of one hundred and ninety-two cases, quotes twenty as being cases in which no anæsthesia was associated with lesions of the gyrus fornicatus or gyrus hippocampi. By his own showing, however, some disturbance of sensation was present in nearly one-third of these cases.

Savill[4] reports left anæsthesia and trophic changes occur-

[1] *Lancet*, ii., 1887. [2] *Medical Week*, vol. i., No. 34.
[3] *Journal of Mental and Nervous Diseases*, Oct., 1888.
[4] BRAIN, 1890.

ring in a case in which a cavity was present beneath the cortex of the right gyrus fornicatus.

Hammond[1] reports a case in which (among other lesions) pressure on the left gyrus fornicatus was associated with anæsthesia of the same side.

If clinical observation, however, lags far in rear of experimental research in this matter, it is satisfactory to note that the direction of the former is in the footsteps of the latter. Thus Horsley and Schäfer,[2] expanding Ferrier's previous conclusions, are of opinion that the gyrus fornicatus represents, in the hemisphere, the area of cutaneous sensibility, and on the clinical side, Mills, after an exhaustive digest of recorded cases,[3] concludes that "no part of the brain is more likely to contain the areas (for cutaneous sensibility, *i.e.*,) than the gyrus fornicatus, the hippocampal gyrus, the precuneus, and the postero-parietal convolutions.

It should be encouraging, too, to remember, that the difficulties which beset a solution of the problem do not differ, in kind or in degree, from those which have hindered the settlement of the question of motor localisation. They arise chiefly from cases, apparently anomalous, in which the specific symptoms are associated with lesions of areas other than those indicated by experiment, and in the second place, from instances, apparently negative in import, in which morbid conditions of the regions indicated by experiment are accompanied by no specific symptoms. Concerning the latter, the phenomena of functional compensation are probably equally valid as explanatory of the apparent anomalies of sensory "negative instances" as they have been shown to be in the case of motor functions. And as regards the first-mentioned difficulty, the accumulation of recorded cases will probably, with a widening knowledge, reveal the explanation of these present-seeming discrepancies.

In the case I have now recorded, the ascertained absence of morbid change from the sensory tracts of the pons, crus, and internal capsule, points to the cortical areas involved, and the parts underlying them as the seat of the sensory disturbance.

[1] *Ib.*, 1891. [2] *Trans. Roy. Soc.*, 1888. [3] BRAIN, 1889.

The location of the softened tract posterior to the internal capsule is such that it would involve the temporal portion of the temporo-occipital cerebellar fibres, which, lying to the outside of the pyramidal fibres in the crusta, are known to radiate outwards and backwards to the temporal and occipital cortex. As these fibres are presumed to be motor, or at least not sensory in function, however,[1] the sensory impairment cannot be associated with their damage. It is reasonable, therefore, to connect the anæsthesia, general and special, with the cortical lesions on the outer and mesial surfaces of the temporal lobe, and with the destruction of the subjacent medullary tissue. Such a conclusion would be in complete accord with the observations of Ferrier and Yeo,[2] who showed that lesions of the hippocampal gyrus, hippocampus, and the neighbourhood of the calcarine fissure, were associated with loss of sensation on the opposite side of the body, and, inasmuch as the hippocampal and uncinate gyri are merely subordinate divisions of the gyrus fornicatus, the conclusion is equally in harmony with the later observations of Horsley and Schäfer.[3]

With reference to the special senses, it is probable that the loss of hearing on the right side was due to intra-cranial pressure on the soft fibres of the right auditory. The right superior T.S. convolution was not affected in its posterior part, otherwise than by being displaced upwards, and by an apparent hypertrophy of connective tissue.

The impairment of taste and smell on the left side were probably contributed to, if not accounted for, by the facial anæsthesia on that side. On the right side, however, the loss may be associated with the damage to the grey and medullary matter of the hippocampal gyrus consequent on the pressure of the tumour.

In considering the almost total amblyopia of the left eye, the difficulty is to assign to each of several morbid conditions present, its legitimate share in the result. There was extreme cloudiness, with, later, ulceration of the cornea.

[1] Gowers, "Nervous Diseases," vol. ii., pp. 29 and 65.

[2] "Functions of Brain," 1886, chap. ix.

[3] *Supra cit.*

It is possible that optic neuritis was also present. I also refer, in this connection, but with diffidence, to possible destruction of fibres of the optic radiation of Gratiolet in the white matter of the temporal lobe, in the area of softening described. I am not aware whether this lesion has ever been associated definitely with crossed amblyopia.

The association of the latter condition with lesions of one angular gyrus has been established by Ferrier,[1] and clinical cases of similar import have been recorded by him, by Grasset,[2] and by Sharkey.[2]

A case of hemianæsthesia, with homonymous hemiopia; in which the softening involved the fibres of the optic radiation, is reported by Beevor;[3] but in his case the area of softening was much more extensive than in mine, extending back to the surface of the occipital lobe.

If the fibres of the optic radiation proceed, in part, to the angular gyrus, as well as to the occipital visual centres, it seems possible that a lesion of small area might affect those fibres only, or chiefly, which are in connection with the angular gyrus of the same side, and so produce crossed amblyopia rather than hemianopia. Although, in my case, the patient's condition rendered futile any attempt at a careful perimetric investigation, yet I am strongly of opinion from the observations made that his visual condition indicated crossed amblyopia rather than hemiopia.

The condition of vision in the right eye was sufficiently accounted for by ascertained optic neuritis.

The patient's mental condition which, on his admission, I thought resembled the condition described as word-deafness, is extremely interesting in connection with Starr's investigations into the pathology of sensory aphasia.[4] In an analysis of fifty cases of sensory aphasia, Starr found that the convolutions most frequently affected were the first and second temporo-sphenoidal. In seven cases pure word-deafness was present, and in all of these the lesion was

[1] *Op. cit.*, p. 287.

[2] *Op. cit.* 290. I have not had an opportunity, however, of referring to the originals.

[3] *Lancet*, May 5, 1895.

[4] Brain, 1889.

limited to the first and second T.S. convolutions, in their posterior two-thirds.

As regards diagnosis, I may say that the only approach to one made during life was the suspicion of a gummatous tumour or tumours, somewhere at the base of the brain. The difficulty, of course, was to harmonise signs which pointed at once to compression of the right oculo-motor, and auditory, with injury to the left trigeminal; and to explain at the same time interference with the sensory tract passing to the left side of the body, without serious implication of the motor fibres. Due weight was perhaps not given to the olfactory indications which might have led to a localising diagnosis of injury in the region of the uncinate and hippocampal lobules, nor were there present to my mind the localising indications afforded by sensory aphasia, according to Starr, and quoted above. Needless to say, no single lesion could be imagined associating all the signs present, and the invocation of multiple lesions did not afford much assistance. It is to be hoped that sufficient clinical material will eventually be accumulated to allow of greater confidence in the localisation of the cerebral sensory areas, and the present case is recorded with a view to that end.

ON DIPHTHERITIC PARALYSIS.

Being an Analysis of the Cases occurring at the Eastern Hospital, Homerton, during the Years 1892 *and* 1893.

BY E. W. GOODALL, M.D.LOND.

Medical Superintendent of the Hospital.

Before entering upon the subject-matter proper of this paper, there are a few preliminary observations which I desire to make. Firstly, with respect to the nature and circumstances of the cases of diphtheria that have been admitted into the hospital during the two years 1892 and 1893: the patients came almost entirely from the eastern and north-eastern districts of London, and were furnished mostly by the lower and lower-middle classes of society (labourers, mechanics, small shopkeepers, clerks, &c.). The severity of the cases, judged by the mortality, was the same each year; the mortality per cent. (calculated according to the Registrar-General's formula) was 35·6 for 1892 and 35·2 for 1893. The cases, both of diphtheria and of paralysis, were consecutive. All cases of disease sent to the hospital certified to be diphtheria, but found to be of another nature, are excluded.

Secondly, with respect to the length of time the patients were detained in the hospital: it has been my practice (and it has been rigidly carried out) not to discharge any child before the end of six weeks from admission, unless the attack of diphtheria began any length of time before admission, which, however, is very exceptional. Adults are detained for six weeks only when the attack is severe or when some complication arises to prevent their discharge; otherwise, they are allowed to leave at the end of four weeks at the earliest. A considerable number of patients have been sent, at the end of three or four weeks, to the Northern (Convalescent) Hospital at Winchmore Hill, where they have

remained for a further period of three or four weeks. A few of these patients have been attacked with paralysis while at Winchmore Hill; Dr. Hume, the medical superintendent of the Northern Hospital, has kindly supplied me with information concerning these cases, and they are included in my list.

Thirdly, nearly all the cases of diphtheria, and still more of paralysis, have come under my own personal observation; but for the bulk of the written notes I am much indebted to the assistant medical officers of the hospital, who have taken much trouble over them.

I have made these remarks in order that others may get some idea of the class of case with which this paper deals, and of the conditions under which the cases have been observed. It will be found that some of my statements differ from those made by other writers on the same subject. But, inasmuch as the experience derived from one hospital may differ widely from that of another, and both from that gained in private or consulting practice, it is not surprising to find even marked difference of statement with respect to what are supposed to be questions of fact, let alone of opinion. The truth can only be arrived at by a combination of experiences.

Number of Cases.—During the years 1892 and 1893, 1,071 cases of primary diphtheria[1] have been under treatment in the hospital, including those remaining at the end of 1891 and excluding those remaining at the end of 1893. Amongst these cases there have been 125 instances of paralysis, an incidence of 11·6 per cent. on the total cases; 17 of the 125 cases proved fatal. It has to be remembered, however, that in diphtheria death, due to the attack itself, takes place fairly early—before, to employ Roger's expression, the patient has time to become paralytic. Hence, a fairer estimate of the frequency of occurrence of paralysis will be obtained if the fatal cases (less, of course, those fatal during paralysis) be subtracted from the total, and the incidence be calculated upon the remainder. There were 362 such fatal cases, so that 709 patients survived to run the risk of becoming paralytic; 125 were so affected—an incidence of 17·6 per cent.

Sex.—Of these 125 patients, 55 were males and 70 females—a proportion of 1 male to 1·27 females. This slight preponderance of females may possibly be accounted for as follows:

[1] As recognised clinically; by primary is meant not secondary to scarlet fever, measles, &c.

more females than males were under treatment—the 1,071 cases being made up of 478 males and 593 females (one male to 1·24 females); moreover, the mortality was higher among males than females—183 of the former and 179 of the latter having died (excluding deaths due to or occurring during paralysis), so that 295 males and 414 females lived to run the risk of paralysis (1 male to 1·4 females); seeing, however, that 55 of these males and 70 of these females did become paralytic, it would seem that males are a little more likely to be so affected than females, for the incidence in the former sex is 18·6 per cent.; in the latter, 16·9.

Age.—The numbers of cases are arranged in the following table according to age:—

Age.	*No. of Cases.*
Between 1 and 2 years	2
„ 2 „ 3 „	7
„ 3 „ 4 „	13
„ 4 „ 5 „	13
„ 5 „ 6 „	17
„ 6 „ 7 „	17
„ 7 „ 8 „	11
„ 8 „ 9 „	8
„ 9 „ 10 „	8
„ 10 „ 11 „	8
„ 11 „ 12 „	6
„ 12 „ 13 „	4
„ 13 „ 14 „	2
„ 14 „ 15 „	3
„ 17 „ 18 „	2
„ 23 „ 24 „	1
„ 26 „ 27 „	1
„ 28 „ 29 „	1
„ 42 „ 43 „	1
	125

Thus the ages of the patients range from one to forty-two years; there were none under one year of age, and only 9 under three and 6 over fifteen years. Most of the patients were, however, under ten years, as will be seen from the following table, in which the cases are arranged in periods of ten years:—

Under 10 years	96
10 to 20 „	25
20 „ 30 „	3
30 „ 40 „	0
40 „ 50 „	1
	125

These figures differ widely from those given by Landouzy in 68 cases quoted by Dr. Gowers in his treatise on "Diseases of the Nervous System" (2nd edition, vol. ii., p. 903). The statements Dr. Gowers makes, apparently in consequence of Landouzy's figures, that "adults furnish a larger proportion of the cases of paralysis than of diphtheria," and that "it would seem that the tendency to paralysis is very slight in early infancy and that it increases the older the sufferer," appear to me to require some modification. No doubt the statements are in accordance with Landouzy's figures, but there must have been some factors in operation causing adults rather than children the subjects of diphtheritic paralysis to be brought under Landouzy's notice. If we arrange the 1,071 cases of diphtheria in ten-yearly age groups with the deaths at the corresponding periods we obtain the following table:—

Under 10 years,	780	of whom	359	died,	15	after paralysis
10 to 20 „	176	„	12	„	1	„
20 to 30 „	73	„	4	„		
30 to 40 „	30	„	2	„		
40 to 50 „	9	„	1	„	1	after paralysis
over 50 „	3	„	1	„		
	1.071		379		17	

Comparing this with a similar table for the cases of paralysis given above we find that of 780 patients under 10 years of age, 96 became paralysed, or 12·3 per cent.; of 176 between 10 and 20 years, 25 became paralysed, or 14·2 per cent.; and of 115 of 20 years of age and upwards, 4 became paralysed, or 3·4 per cent. But the fairest way to get at the incidence of paralysis is (as has been before stated) to subtract from the total cases of diphtheria all those that proved fatal, with the exception of those fatal after the onset of paralysis. If this be done we have 436 cases under 10 years of age, of whom 96 became paralysed, or 22 per cent.; between 10 and 20 years of age there are 165 cases, of whom 25 became paralysed, or 15·1 per cent.; above 20 years of age there are 108 cases, of whom 4 became paralysed, or 3·7 per cent. Again, of 125 cases of diphtheritic paralysis 96 or 76·8 per cent. were children under 10 years of age; 25, or 20 per cent. were aged from 10 to 20 years, and 4, or 3·2 per cent. were more than 20 years old; and of the 1,071 cases of diphtheria, amongst which the cases of paralysis arose, 73 per cent. were under 10; 16 per cent. between 10 and 20, and 11 per cent. were above 20. On the question of age incidence De Gassicourt's treatise on the "Diseases of Childhood," and the work on "Peripheral Neuritis" by the late Dr. Ross and Dr. Bury, should be consulted.

The following table gives these figures concisely :—

Age.	Number of cases of diphtheria.	Deaths, excluding deaths after paralysis.	Number of cases *minus* number of deaths.	Number of cases of paralysis.	Percentage of cases of paralysis on cases *minus* deaths.	Percentage of cases of paralysis on total cases.	Number of deaths after paralysis.
Under 10	780	344	436	96	22·0	12·3	15
10 to 20	176	11	165	25	15·1	14·2	1
20 to 30	73	4					0
30 to 40	30	2	108	4	3·7	3·4	0
40 to 50	9	0					1
50 and upwards	3	1					0
Total ..	1071	362	709	125	17·6	11·6	17

Date of onset of Paralytic Symptoms.—In 101 of the cases the date of commencement of the attack of diphtheria is definitely stated as well as the date of the first appearance of paralytic symptoms. I find that the seventh is the earliest day upon which such symptoms have been observed, and the forty-ninth is the latest; taking the weeks in order, in 2 cases paralysis set in during the first week, in both cases on the seventh day, in 22 cases during the second week, in 28 during the third week, in 22 during the fourth week, in 15 during the fifth week, in 8 during the sixth week, and in 4 during the seventh week.

Usually the membrane or exudation clears off completely before the paralytic symptoms set in; but not always. I have notes of 84 cases in which the length of time during which exudation remained upon the fauces or in the nasal passages was recorded; in 10 of these cases the exudation had not disappeared when the paralysis commenced (for instances, see Cases 5 and 21).

Muscles first affected.—With respect to the parts in which the symptoms of paralysis first show themselves, in 83 cases the palate was first affected, in 20 the ciliary muscles, in 5 the palate and ciliary muscles, in 4 the palate and lower extremities, in 3 the lower extremities, in 2 the muscles of the pharynx, and in 2 the respiratory muscles. In the remaining 6 cases the parts first affected were as follows, there being one instance of each: palate

and upper extremities; ciliary muscles and lower extremities; pharynx and lower extremities; lower extremities and ocular muscles; muscles of trunk and neck; muscles of trunk and extremities. It will thus be seen that in a large proportion of the cases (66·4 per cent.) the palate alone is the first part to be affected; while either alone or in combination with some other muscles it is the first part to suffer in 74·4 per cent. of the cases.

Extent of Paralysis.—In 28 cases the paralysis remained limited to the palate; in 17 to the ciliary muscles[1]; in 11 to the palate and ciliary muscles; in 6 to the palate and lower extremities, including one case in which the vagus was probably also involved (Case 14); in 3 to the palate and one or more ocular muscles; and in 1 to the respiratory muscles including the larynx; in this case too the upper extremities were slightly affected (see Case 10). Whether the vagus or its branches ever become affected alone is a question I shall discuss later. From the above list of cases it will be seen that in 66 of the 125 cases the paralysis was limited in extent, that is, in 52·8 per cent. It is very likely that a few cases of ciliary paralysis have been undetected, many of the patients being young children. All who are old enough to know their letters are tested regularly with Jäger's types. Myopia also conceals slight ciliary paralysis.

In 16 cases the paralysis was generalised.

Frequency with which certain Muscles or Groups of Muscles are affected.—In 102 cases, the palate was paralysed; in 56, the ciliary muscles; in 52, the lower extremities; in 26, one or more of the external ocular muscles; in 21, the upper extremities; and in at least 11, the muscles of the larynx.

In 19 cases, difficulty in swallowing was a marked symptom; but this number does not by any means represent all the cases in which the pharyngeal muscles were paralysed. In all cases where there is marked regurgitation of fluids through the nose, the œsophageal tube is employed for feeding; hence subsequent pharyngeal paralysis is undetected.

Of the 26 cases in which the *external ocular muscles* were affected, in 14 the notes only state the presence of squint, without specifying the exact muscle or muscles paralysed. In 7 of the remaining 12 cases, one external rectus (usually the left) was paralysed; in 3, both external recti; and in 2, most of the muscles of the eyeballs (Case 12 was one of these). In none of

[1] No attempt has been made in any of the cases to determine whether only one or both ciliary muscles were affected.

the cases were the internal recti affected apart from the external. In Case 12 there was left ptosis.

In 10 cases there was evidence of paralysis, more or less complete, of the *diaphragm;* 4 of these were fatal cases.

In 11 cases there was what I have taken to be evidence of paralysis of the *adductors of the vocal cords*, viz.: aphonia, with a toneless and ineffectual cough. Five of these cases were fatal. In 6, the diaphragm was also affected.

In 3 cases the *abductors of the larynx* appeared to be paralysed. The following are the notes of the most marked example:—

Case 1.—Annie G., aged 3½ years, admitted on May 28, having been ill since the day before. There was a little deposit on the right tonsil, and slight laryngeal obstruction. Seven hours after admission tracheotomy was performed, the laryngeal obstruction having become much worse. Membrane was removed from the trachea, and the relief afforded was not immediately complete; but the next day the patient was very comfortable. On May 31, a good deal of membrane was coughed up through the tube. On June 1, the tonsil was clear of deposit.

There was albuminuria on June 3, and this continued till August 7.

On June 16, it was noted that the child could not drink, as violent coughing was set up. Since the operation she had been fed with a tube passed into the stomach. The tracheotomy wound was quite healed by June 23, and by June 27 the child was able to take nourishment in the usual way without any difficulty. On July 2, the child was unable to sit up in bed, and there was a slight squint.

July 3.—"Some cough during the morning. At 11 a.m., sudden cyanosis and dyspnœa. Much recession of supra-clavicular spaces, but not much of chest wall." This attack of dyspnœa was so urgent that the assistant medical officer, Mr. H. E. Mann, who was called to the patient, had to perform tracheotomy, and insert a tube without delay. Complete relief was afforded by this measure. There was no membrane in the trachea.

July 7.—"The patient is quite unable to breathe through the larynx."

July 10.—"The tube was omitted yesterday. To-day, the child is breathing through the mouth without difficulty." By July 14, the wound was healed. On August 1, it was noted that the paralysis was improving; the patient could stand without assistance; drink did not return through the nose; the voice was still nasal, though not so much as before.

August 8.—"The patient is improving, but there is still some stridulous breathing during sleep." She remained in about the same condition till August 26, when the following note was made:—"Patient had an attack of urgent dyspnœa early this morning, which appeared to be spasmodic, and during which she became very blue. On the acute paroxysm passing off, considerable stridor remained, and some recession of the chest walls." Further dyspnœa set in later. Intubation of the larynx was then performed, and relief afforded. The child passed a comfortable night. The tube was removed twenty-four hours after insertion; the child could breathe easily. She never had any further respiratory trouble, except on the night of September 2, when the breathing was observed to be harsh. She left the hospital well on October 8.

No note has been made about the character of the voice, but I distinctly remember that it was gruff before the second tracheotomy, and during the period between the second tracheotomy and the intubation, and for some time after. There was no evidence that the secondary respiratory difficulties were due to any reformation of membrane. There was no vomiting or cardiac irregularity.

In none of the cases was *facial paralysis*, paralysis of the *tongue*, or of the *sphincters of the bladder* or *rectum* observed.

Cardiac Irregularity.—In 29 of the cases it was noted that at some time or other during the paralysis there was irregularity of the cardiac rhythm, usually with increased frequency of beat. This condition was met with chiefly in the severer cases of paralysis, but sometimes it occurred in connection with very slight and limited palsy, such as of the palate or ciliary muscles. Indeed, cardiac irregularity occurs after diphtheria quite apart from paralysis. The duration of the cardiac disturbance may be from a few days to several weeks. Sometimes there is dilatation of the heart.

Respiratory Irregularity.—Very occasionally the respiratory rhythm is observed to be altered. It was so in four cases, of which two were fatal—Cases 10 and 11, and perhaps, too, Cases 6 and 14 (see also Case 24). The following are the notes of the two cases which recovered:—

Case 2.—Lillie M., aged 3 years, sister of Case 8, admitted July 10, having been ill since the 8th. There was much membrane on the palate and tonsils, which did not clear off for several days. There was a faint trace of albumen in the urine. The amount of albumen increased so that by July 22 the urine was loaded. On this date the quantity of urine passed in the twenty-four hours was only one ounce, and the quantity continued to be

scanty for the following four or five weeks. The urine was not free from albumen till October 18. The first symptom of paralysis was a nasal voice on July 26. Gradually there supervened cardiac irregularity, and paralysis of the upper and lower extremities, of the trunk, of the left external rectus oculi, and of the ciliary muscles. On August 27, she had attacks of irregular breathing, the respirations occurring in sighs and gasps. The diaphragm appeared to act very imperfectly. The attacks of irregular breathing were followed by periods of quiet, regular breathing, in which the respiratory rate was 32 per minute, and the pulse 128. After this, the patient gradually improved, and was well by the end of September.

Case 3.—Sidney S., aged 2½ years, admitted March 11, having been ill since the 6th. There was membrane on the soft palate, which persisted for a week after admission; there was also a discharge from the nose and slight laryngeal symptoms, which were present till March 23. There was albumen on one occasion only—March 17. Paralysis set in on March 24, with deficient movement of the thorax; next day it was observed that he had symptoms such as I have above ascribed to paralysis of the adductors of the vocal cords. Pulse, 142; respirations, 60. On March 26, the breathing was irregular, respiration consisting of a number of rapid, followed by a series of slower, respiratory acts. The average rate was 40 per minute; pulse, 140. On the 28th, the breathing was still irregular, but less so than before. Subsequently, the trunk and neck muscles became partially paralysed, and also those of the upper extremities, and, to a slight extent, those of the lower. The intercostal muscles and diaphragm also became affected, and still acted imperfectly as late as April 28. By the beginning of June paralytic symptoms had quite disappeared.

It will be observed that in both these cases as well as in the fatal cases (numbers 10 and 11) and Case 24 related hereafter, irregularity of respiratory rhythm accompanied paralysis of the respiratory muscles, slight in Case 24.

Sensory Disturbances are, perhaps, more common than is supposed. Those most frequently met with are sensations of "pins and needles" or of tingling in the fingers and toes, and numbness of the same parts. In such cases careful testing will elicit the presence of partial loss of ordinary sensation. In the case of children, however, it is very difficult to be certain about slight sensory changes. As most of my patients were children, I have notes of very few cases of sensory disturbance. The following is an account of the best example :—

Case 4.—Alfred H., aged 28 years, admitted on July 11, having had a sore throat since July 9. There was much swelling of the tonsils and palate; the tonsils were covered with thin membrane. The breath was fœtid. During the next two or three days the membrane spread to the palate and uvula, and there was a discharge from the nose. The fauces were not clear till July 22. There was a good deal of albumen in the urine from the day of admission till the end of July. The urine was not free from albumen till August 24.

The first symptom of paralysis was inability to read small print on August 2. On that date the knee-jerks were brisk. The next was a feeling of numbness in the tongue, first noticed on August 15; this lasted for about a week. Towards the end of the third week in August other sensory and motor disturbances appeared which progressed during the next fortnight. On September 8, the following note was made:—"During the past fortnight the patient has complained of weakness and numbness of the left arm. To-day there is a marked deficiency in the power of the muscles of the left forearm and hand; also slight deficiency in the muscles of the upper arm. The power of the left little finger is very weak, that of the ring finger less so, and still less that of the remaining fingers and the thumb. But the muscles of all these digits are decidedly weak when compared with those on the right side. There is loss of common sensation on the palmar aspect of the left little and ring fingers, and this condition extends up on to the corresponding part of the palm and forearm. On the dorsal aspect sensation is only impaired. Both knee-jerks are present. There is no weakness of the lower extremities. The elbow and wrist-jerks on each side are present but feeble. No anæsthesia of right upper or of lower extremities. No motor paralysis of right arm. He can read J. 4. The patient has in fact slight ciliary paralysis, and partial left brachial monoplegia."

During the next few days the ciliary paralysis disappeared. But the right upper extremity became partially paralysed and the man complained of numbness of the feet. On September 20, it was noticed that the knee-jerks were absent, and his gait was ataxic. He complained of pain in the legs below the knees, especially at night. There was still numbness of the feet. The grasp of the right hand was very weak, that of the left *nil*. The patient then gradually improved, and left the hospital well on October 23.

Fatal Cases.—An analysis of the 17 fatal cases shows that in 13 the cause of death was to be put down to paralysis. Six of

these cases proved fatal from cardiac failure; 4 from respiratory paralysis; 2 from persistent vomiting and cardiac failure, and 1 from convulsions. In these 13 cases death took place at times varying from twenty-nine to sixty-two days after the onset of the attack of diphtheria, and on the third to the thirty-fourth day of the paralysis.

In 2 of the remaining 4 cases death was due to the severity of the attack of diphtheria, and took place on the fifteenth and nineteenth day of the disease, and the fourth and sixth of the paralysis, which was limited to the palate in each case. In the third case death was due to a relapse of diphtheria occurring when the patient was recovering from paralysis; while in the fourth case, Case 21, death did not supervene till the thirty-eighth day of the disease, yet membrane persisted upon the fauces, and death was probably due to the direct effects of the poison of diphtheria. Fifteen of the 17 deaths were in patients under 10 years of age.

Albuminuria.—In 122 out of the 125 cases a record has been made as to the presence or absence of albumen in the urine. In only 12 cases has albumen been absent. In 2 out of the 110 cases in which albumen was present, no note has been made as to the length of time during which the albuminuria lasted. In 20 of the remaining 108 cases the duration of the albuminuria was less than one week; in 88 it was for a period varying from one to thirteen weeks. The amount of albumen present was very variable, both in different cases, and in the same case at different times.

Duration of Paralysis (excluding the fatal cases).—In 96 cases it was noted with tolerable accuracy how long the attack of paralysis lasted. In the following table the results of the analysis of the notes in this respect are shown:—

Under one week	..	..	..	..	..	6 cases
Between 1 and 2 weeks	..	..	..	..	1 case
„ 2 „ 3 „	..	..	..	..	3 cases
„ 3 „ 4 „	..	..	..	..	5 „
„ 4 „ 5 „	..	..	..	..	7 „
„ 5 „ 6 „	..	..	..	..	9 „
„ 6 „ 7 „	..	..	..	..	15 „
„ 7 „ 8 „	..	..	..	..	21 „
„ 8 „ 9 „	..	..	..	..	7 „
„ 9 „ 10 „	..	..	..	..	5 „
„ 10 „ 11 „	..	..	..	..	8 „
„ 11 „ 12 „	..	..	..	..	4 „
„ 12 „ 13 „	..	..	..	..	2 „
„ 13 „ 14 „	..	..	..	..	2 „
„ 14 „ 15 „	..	..	..	..	1 case

In none of the cases was any permanent paralysis left behind. There is no intermediate result between death and recovery.

With respect to the *connection between the amount of exudation upon the fauces and the subsequent paralysis*, the cases have been divided into four groups:—

(1) Those in which there was no exudation—3 in number.
(2) Those in which the amount was slight—24.
(3) Those in which there was a moderate amount—45.
(4) Those in which there was much exudation—53.

These observations, of course, only apply to the condition of the fauces as seen after admission to the hospital. It is quite possible, for instance, that in the 3 cases in which no exudation was observed on or after admission, that it had been present before admission. In 2 of these 3 cases there was superficial ulceration of the soft palate, which was slow in healing.

It is commonly believed that paralysis is as likely to follow a mild as a severe attack of diphtheria. In fact, no less an authority than Henoch says:—"Diphtheritic paralysis occurs most commonly after the milder attacks of the disease."[1] So also Dr. Gowers:—"Nor does the severity of the attack of diphtheria determine the occurrence of palsy; it is equally likely to follow attacks that are severe and those that are trifling."[2] With this opinion, however, I do not agree. I will not, of course, deny that paralysis may follow a mild attack, for I have myself seen it do so; but my experience is that the more severe the attack of diphtheria, the greater the likelihood of subsequent paralysis. In diphtheria, severity of attack depends very largely upon extent and persistence of the local exudation—membranous or otherwise. The amount and duration of albumen in the urine give a fair idea of the degree of poisoning by the toxic products of the bacillus diphtheriæ. A reference to the paragraph headed "*Albuminuria*" will show that in, at least, 110 of the 125 cases of paralysis, there was albuminuria, 88·0 per cent.; while in 88 of the cases, 70·4 per cent., the albuminuria lasted for a week or more. Albuminuria was found in about 30 per cent. of the 1,071 cases of diphtheria, amongst which the cases of paralysis under consideration occurred, so that the paralytic cases show a larger proportion having albuminuria. Again, a reference to the table just given will show that in 27, or 21·6 per

[1] "Lectures on Children's Diseases," *Syd. Soc. Trans.*, vol. ii., p. 300.
[2] *Loc. cit.*, p. 904.

cent. of the cases of paralysis, there had been little or no local exudation; in 45, or 36·0 per cent., there had been a moderate amount; and in 53, or 42·4 per cent., there had been much; that is, in nearly half the cases of paralysis there was much local exudation, in spite of the fact that it is just the cases with much exudation that are fatal during the attack of diphtheria, at a time too early for paralysis to have supervened.

Further, I have analysed, in more detail, the severe and the mild cases of paralysis. There were 22 severe cases; 13 of them were, in fact, fatal from the paralysis. In 10 of these 22 cases there was much albumen in the urine; in 10 there was a moderate amount; in 2 there was a trace on one occasion only; and in none was the urine always free from albumen. With the two exceptions mentioned, albumen was present for a period of eleven to one hundred and four days' duration.

With respect to the amount of exudation present during the primary attack of diphtheria in these 22 cases, there was much in 11 cases, a moderate amount in 7, a slight amount in 3, and none in 1. In this last case there was ulceration of the soft palate, which was very slow to heal; and my belief, when I saw the child soon after admission, was that there had been a good deal of membrane before she was sent to the hospital, especially, too, as there was much albumen persisting for at least seventy-seven days. I may also add that there was in 11 out of the 22 cases a discharge from the nose, pointing to the presence of membrane in the nasal passages; and in 5 cases there was croup.

Contrast these figures with the following, which are drawn from an analysis of those cases of paralysis in which only the palate or the ciliary muscles were affected, that is to say, the very mildest cases of paralysis. Excluding 2 fatal cases in which the patients died of the severity of the disease during the primary attack, and in which there was hardly time for the paralysis to have advanced beyond the palate, there were 43 such mild cases. In 7 of these there was much albumen, in 21 there was a trace or a cloud, in 7 there was albumen on one occasion only, and in 8 there was never any albumen. The extremes of times of duration of albuminuria were five and seventy-six days. With respect to the amount of exudation in these 43 cases, the amount was slight in 12, moderate in 18, and much in 13. There was a discharge from the nose in 7 cases, and croup in 3 others.

In a tabular form these figures can be placed as follows:—

Albuminuria.	Severe cases of paralysis.	Mild cases of paralysis.	Faucial exudation.	Severe cases of paralysis.	Mild cases of paralysis.
Much	10	7	Much	11	13
Moderate amount	10	21	Moderate ..	7	18
Present once only	2	7	Slight	3	12
None	0	8	None	1	0
	22	43		22	43

The observations of Cadet de Gassicourt on this subject do not differ very widely from mine. They will be found on page 342 of the third volume of his *Traité Clinique des Maladies de l'Enfance* (deuxième édition), in the admirable chapters which he devotes to diphtheria. De Gassicourt excludes all his cases of diphtheria which were fatal from croup. This leaves him 425 cases, which he divides into three groups: first, the benign cases; second, the severe, toxic and hypertoxic cases; and third, the cases of croup that recovered either with or without operation.

In the first group there were 155 cases with 28 of paralysis, 18 per cent. In the second group there were 135 cases, with 30 of paralysis, 22·2 per cent. In the third group there were 135 cases with 43 of paralysis, 31·8 per cent. The number of fatal paralytic cases in each group was 2, 10 and 8. In a short discussion of these figures de Gassicourt states that he is surprised at the difference between the numbers of cases occurring in the second and third groups; he thinks the explanation to lie in the fact that many of the 135 severe cases of diphtheria died of the malignity of the disease before they had time to become paralytic; but he does not give the actual number who so died.

If de Gassicourt's cases of paralysis are cast into the same form of table as mine it will be seen that out of 101 cases, in 28 the primary attack was benign, in 30 severe, and in 43 there was croup; from the context one gathers that these latter cases were severe or moderately severe cases of faucial, laryngeal and in some instances tracheal diphtheria.

My cases of croup in diphtheria that recovered with or without an operation differ, however, considerably from those enumerated by de Gassicourt. I have had 54 such during the two years 1892 and 1893; 13 of these patients subsequently developed paralysis (24 per cent.), of whom 1 died.

With these facts before us I do not think it can be maintained that paralysis follows a mild attack of diphtheria equally as often as a severe.

The following are brief notes of the 17 fatal cases:—

Case 5.—Severe diphtheria, faucial and nasal; generalised paralysis; sudden death from cardiac failure. F., aged 9; sore throat, May 31; admitted on June 5, with the fauces in a very foul condition; there was a sanguineous discharge from the nose, and the child seemed extremely ill. On June 13, the fauces were cleaner; there was still what seemed to be a film of membrane on an ulcerated surface; on the 15th no membrane could be seen, only the ulceration. On June 30, and July 3, membrane was syringed out of the nose. From June 14 to 19th, a well marked papular eruption was to be seen on the skin; on June 22, there was cardiac dilatation; and on July 1, otorrhœa. On this date, too, paralytic symptoms were first observed, the patient being unable to raise her head from the pillow; by July 3, there was marked paresis of the muscles of the trunk and extremities; the voice was nasal; the knee-jerks were absent; the abdominal muscles acted. There was difficulty in swallowing, and paralysis of the ciliary muscles. Pulse 160; respirations 22. The patient remained in the same state till 6.30 p.m., on the same day when she died very suddenly, having been talking and laughing with the nurse almost up to the very moment of death.

Case 6.—Slight diphtheria, faucial; slight croup, generalised paralysis, including laryngeal and respiratory muscles; death by cardiac failure. M., aged 5; sore throat, April 19; admitted the next day with a patch of membrane on the left tonsil; by the 27th the membrane had disappeared. There was a cloud of albumen in the urine from April 28 to May 18, and once again on June 2. There was slight croup from April 24 to 27; the patient was troubled with vomiting from April 27 to 30. Soon after he was admitted the cervical glands became enlarged and subsequently they suppurated. The first symptom of paralysis was a nasal voice on May 17. On May 21 there were aphonia, loss of cough (adductor paralysis of larynx), paresis of the arms and legs; no knee-jerks; vomiting; and irregularity of the heart's action. On May 26, he surreptitiously obtained a portion of an orange and nearly choked whilst eating it. On the 30th it was noted that the thorax expanded badly, that the diaphragm was weakened, and that the child could not sit up or even lift his head off the pillow. At 3 a.m., on June 1, there was dyspnœa; the patient became of a bad colour; pulse 120 and regular. At 1 p.m., he

was collapsed and there was no dyspnœa; his colour was better. At 11 p.m., he was seized with convulsive movements of the face and arms; the pupils became dilated. These convulsions did not last long. There was some improvement in the patient's condition during the next two days; but he died suddenly from cardiac failure at 5.30 p.m., on June 3.

Case 7.—*Severe attack of diphtheria; paralysis of palate and extremities; death by cardiac failure.* F., aged 4; sore throat on September 8; admitted on September 9, with patches of membrane on the tonsils and fauces; child very ill. There was still membrane on the 11th; the urine contained albumen and continued to do so up till death. The cervical glands were swollen. On October 6, there was a nasal voice. Then the muscles of the extremities became affected. On October 11, there was strabismus; next day there was slight weakness of the diaphragm, and also icterus. The patient died of cardiac failure on October 12.

Case 8.—*Severe diphtheria, faucial and nasal; generalised paralysis; death by cardiac failure.* F., aged 6. Sore throat on July 7; admitted on July 8, with much black tough membrane covering the fauces. By July 13, the fauces were clear. On admission there was rather more than a trace of albumen in the urine; on July 11, the urine was loaded with albumen and it continued to contain much albumen till death. There was also a fœtid discharge from the nose, and cervical lymphadenitis. On July 21, the voice was slightly nasal, the knee-jerks could be obtained but were not well marked, and the pulse 108 and irregular. The patient could read small print. On July 28, the heart was dilated. By August 5, the patient could not stand nor even raise herself in bed. On the 6th, there were no knee-jerks: there was ocular and ciliary palsy. On the 8th, the paralysis was more or less generalised; the patient could not swallow properly. She got worse and died of heart failure on August 11.

Case 9.—*Severe diphtheria, faucial and (?) nasal; generalised paralysis; death by cardiac failure.* M., aged 5. Sore throat on June 4; admitted June 6, with much deposit on the tonsils and palate. The fauces were clear on June 13. There was a discharge from the nose and ears and much swelling of the cervical glands. On June 8, there was rather more than a trace of albumen in the urine, but the quantity increased so that on the 23rd the urine was loaded, and at the same time the amount of urine passed in the twenty-four hours was scanty. This state of affairs continued up till death, except that for a day or two before

the end there was a slight improvement in the condition of the urine. Up to June 21, the knee-jerks could occasionally be obtained, but after that date they were absent. Paralysis of the palate was first noticed on June 22; on the 25th there was weakness of the lower extremities. Then gradually paralysis appeared in other parts so that on July 13 there were nasal voice and palsy of the muscles of the upper and lower extremities, the trunk and the neck. On July 14, the patient had to be fed with a tube as he could not swallow. He died the same evening of cardiac failure. As early as June 17, there was evidence of cardiac dilatation; after that date the pulse rate varied from 100 to 124 per minute, and the heart's action was irregular. On the day of death the rate was 172.

Case 10.—*Slight diphtheria, faucial and (?) nasal; paralysis of respiration.* M., aged 3½. Sore throat on February 3; admitted February 7, with membrane on both tonsils. On February 13, the fauces were clear, but on the 14th there was again a small patch on the pharynx. There was albumen in the urine from the day of admission till death. There was also a discharge from the nose, and a cervical abscess which was opened on March 3. Paralysis set in on March 5, with sudden acute abdominal pain; it was observed that the movement of the chest walls was much impaired and the cough was very inefficient, the cause in this case being probably paralysis of the adductors of the larynx. Next day the breathing was almost entirely abdominal; coughing —or rather attempts at coughing—were set up when the child took liquids. In the evening the respirations became of a sighing character and occurred in groups of three with a pause between each group. On March 7, the grasp was feeble; on March 10, the patient could not cough and at times he was of a very bad colour. He died the same day of respiratory paralysis.

Case 11.—*Moderately severe diphtheria, faucial and (?) nasal; paralysis of palate, lower extremities, left external rectus; death from paralysis of respiratory muscles.* M., aged 3. Sore throat January 3; admitted January 6, with tonsils swollen and covered with membrane, which persisted till January 17, and after its disappearance there was ulceration. The urine contained a trace of albumen from January 9 to February 7; and there was a discharge from the nose. On February 8 there was ataxia and some loss of power in the lower extremities; there were no knee-jerks; the palate was slightly affected. On February 9 there was paresis of the left external rectus; February 10, marked nasal voice. February 11, there were attacks of irregular breathing, three res-

pirations taking place in quick succession and then a pause; the pulse was very frequent. On February 13 the character of the breathing had altered; it was quite regular in rhythm, but a long inspiration was followed by a short expiration. In the evening it was noted that there was some paralysis of the muscles of respiration. This breathing continued till death, which took place at 9 a.m. on February 14; the diaphragm became paralysed before death, and there were twitchings of the muscles of the face and arms. Death was due to respiratory paralysis.

Case 12.—*Severe diphtheria, faucial; ocular paralysis; and of upper limbs; death from respiratory palsy.* F., aged 5. Sore throat on July 1; admitted July 5, with much membrane on the fauces, which were clear by July 14. There was rather more than a trace of albumen in the urine; the amount of albumen somewhat increased up to July 14; then it lessened and on August 14 the urine was free from albumen. On July 21 the voice became nasal and there was ciliary paralysis. July 26, patient pale and listless; knee-jerks present. Pulse 88. August 5, squint and diplopia; ciliary paralysis worse. On August 9 the patient could not stand; there was left external strabismus and ptosis on the left side; both the internal recti were weak; the eyeballs did not converge for near vision and there was also loss of the up and down movements. On August 11 the condition was the same, but it was observed in addition that the pupils were moderately dilated; the right reacted feebly to light, but the left did not; neither reacted to accommodation; the upper extremities were partly paralysed. Common sensation was tested and found normal. On August 13 the paresis had increased; the patient could swallow. Pulse 128. August 18, very prostrate. Pulse 80. On August 20 there was frequent cough; and the next day it was noted that there was deficient expansion of the thorax and difficulty in deglutition. The respiratory palsy increased and proved fatal at 1 a.m. on August 22.

Case 13.—*Moderately severe diphtheria, faucial and (?) nasal; paralysis of lower extremities, palate and ciliary muscles; death from respiratory paralysis.* F., aged 4. Sore throat May 10; admitted May 11, with deposit on both tonsils; the deposit had increased in amount and become membranous by May 13; it then gradually disappeared and on May 21 the fauces seemed normal. There was much albumen in the urine on admission and up till death. There was also a discharge from the nose. On May 28, it was observed that the knee-jerks were slight and the patient could not stand; pulse frequent; on May 31 the voice was nasal,

and next day it was nasal to such a degree that the patient could hardly be understood. On June 3 the ciliary muscles became paralysed; on June 6 there was evidence of slight cardiac dilatation and of weakness of the muscles of the back. On June 15 the heart was still more dilated; there was a systolic apical murmur and the action was irregular. June 19, heart's action "tumbling" in character; pulse 180. The respirations were very shallow; the patient was unable to cough. In the evening of this day it was noted that there was cyanosis and the patient was "gasping for breath like a newly-caught fish." The diaphragm appeared to be acting, but the intercostal and laryngeal muscles were paralysed; there was still a nasal voice and ciliary paralysis. Pupils dilated. The patient died soon after this note was made.

Case 14.—*Moderately severe attack of diphtheria, faucial and (?) nasal; paralysis of palate and lower extremities; death by cardiac failure.* F., aged 6. Sore throat on May 17; admitted May 19, with a thick covering of membrane on the tonsils and uvula; by the 25th the membrane had disappeared. On admission the urine contained a trace of albumen, which continued up to June 16. The cervical glands were enlarged and there was a discharge from the nose. On June 19 there was considerable difficulty in walking; the voice was slightly nasal; the knee-jerks were not to be obtained, and the heart's action was frequent and irregular. On June 26 the knee-jerks could be obtained. On July 1, in the morning the patient was worse; the heart's action was very frequent and irregular; there was vomiting; no albuminuria. In the afternoon the heart's action was regular, 128; respiration 44; the respirations were laboured, and the colour bad. The child died of cardiac failure at 5 a.m., on July 2.

Case 15.—*Severe diphtheria, faucial and laryngeal; tracheotomy; generalised paralysis; attacks of vomiting and syncope; death by cardiac failure.* M., aged 6. Sore throat on March 19; admitted the same day with much membrane upon the fauces, which increased in amount during the next five days. On March 31, there was much albumen in the urine; the amount then diminished and after April 10 the urine was normal. There was croup which necessitated tracheotomy on March 24. From this the patient completely recovered, and was allowed to get up on April 10. On April 29, there was paralysis of the palate and weakness of the right arm; the knee-jerks could not be obtained. Afterwards there was more or less generalised paresis; on May 3, attacks of vomiting and syncope commenced which recurred

occasionally during the next few days and in one of which the patient expired early in the morning of May 8.

Case 16.—*Moderately severe diphtheria, faucial; nephritis; paralysis of palate, extremities and larynx; constant vomiting; death by cardiac failure.* F., aged 42. Sore throat June 28; admitted July 3; with much membrane on the fauces. The membrane had gone by July 8, but there was ulceration of the soft palate and uvula which did not heal up till July 23. It was not possible to examine the urine till July 6, and it was then found to be loaded with albumen and to be smoky, which it continued to be up to July 11. There were also casts. The condition of the urine pointed to an acute nephritis. After July 20, the amount of albumen lessened, and on July 27 there was rather more than a trace, which continued; on August 29 there was a faint trace. From July 13 to 18th the patient was troubled with pain in the chest, to account for which no physical signs were detected; there was also occasional vomiting; pulse 60 to 64; heart intermittent. On July 31 there was a return of fluids through the nose; on August 6 the voice became nasal; on the 19th there was paresis of the upper and lower limbs and ataxia; no jerks could be obtained either in the upper or lower extremities. August 25, vomiting; cough after taking food; pulse 96. During the following night there was vomiting and abdominal pain with collapse, all coming on rather suddenly. Pulse 90; the action and sounds of the heart were normal the next morning. The patient was fed by nutrient enemata on account of the constant vomiting. On August 27 she was almost voiceless; there was frequent vomiting without any feeling of nausea; pulse 96, regular. On August 28 the patient was quite helpless; there was almost complete loss of power in the lower, and marked paresis of the upper extremities; vomiting continued. Pulse 96; heart normal and regular. The patient got weaker and died quietly shortly before 6 a.m., on August 29. Only a partial *post-mortem* examination was permitted. The abdominal organs were found to be quite normal to the naked eye.

Case 17.—*Severe diphtheria, faucial and nasal; paralysis of palate, intercostals and laryngeal muscles; death in convulsions.* M., aged 4. Sore throat February 15; admitted February 17 with much membrane spread over the fauces. Membrane was still present on February 24; on February 28 there was a trace of albumen in the urine; there was a discharge from the nose; later also diarrhœa and a cervical abscess. On March 13 there was paralysis of the palate; on the 15th there was weakness of the

intercostals and adductors of the larynx; the patient was seized with convulsions during the day and speedily died.

Case 18.—*Severe diphtheria, faucial and nasal; paralysis of palate; death from severity of attack.* F., aged 10. Sore throat November 13; admitted November 15, with very much membrane on the fauces, which had much decreased but not quite disappeared at the time of death. There was albuminuria, and a discharge from the nose. The patient was very ill. On November 24 there was a decidedly nasal voice; heart normal. The patient got weaker, and died from the effects of the poison of diphtheria on November 28.

Case 19.—*Severe diphtheria, faucial and nasal; paralysis of palate; death from severity of disease.* M., aged 9. Sore throat on May 10; admitted May 13 with much swelling of the fauces and much membrane. The membrane gradually disappeared by May 24. There was a good deal of albumen in the urine from the day of admission to the day of death. There was epistaxis on the 16th and 17th. On May 23 the patient had a nasal voice. No further symptom of paralysis occurred till the morning of May 29, when there were symptoms of cardiac failure; no vomiting. The patient died of cardiac failure at 7.30 p.m. the same evening.

Case 20.—*Moderately severe diphtheria, faucial with slight croup; generalised paralysis; relapse of diphtheria, faucial and laryngeal; tracheotomy, death.* F., aged 2½. Sore throat on September 25; admitted on September 28 with membrane on the tonsils and uvula; there was still a little membrane on the uvula on September 30; there was a cloud of albumen in the urine on September 30; albuminuria existed till November 20. When the patient was admitted there was slight croup. Paralysis of the palate was observed on October 6; by November 11 there was more or less generalised paresis with strabismus. The child however had much improved when on December 28 a relapse of diphtheria occurred; the membranous formation spread to the larynx and trachea; the child was tracheotomised with only temporary relief, and death took place on January 2.

Case 21.—*Prolonged diphtheria; faucial, laryngeal and nasal; paralysis of palate, ciliary muscle and one external rectus; death from direct effects of diphtheritic poison.* M., aged 6. Admitted July 30; there was swelling of the tonsils, with membrane on the left tonsil and on the uvula. On August 1 there was no membrane but slight ulceration in its place. On August 9 there were again some specks of deposit on the fauces; these cleared off, but again on the 20th the fauces became swollen, and there was membrane on

the right tonsil; the next day there was some on the left as well. On August 25 there was a discharge from the nose and enlargement of the glands in the neck; on the 26th slight croup appeared, and next day the child coughed up a small piece of membrane. There was still membrane on both tonsils on August 31. There was a cloud of albumen in the urine when the patient was admitted; the amount quickly increased, and for some days before death the urine was loaded. Symptoms of paralysis set in on August 3 in the palate, the ciliary muscles being also slightly affected. On August 6, 15 and 19, it was noticed that the knee-jerks were exaggerated; by the last date the palsy of the palate and ciliary muscle had become more pronounced. On August 23 the knee-jerks had almost disappeared. On September 5 the left external rectus was paralysed. The patient died on September 6, most probably from the direct effects of the diphtheritic poison.

An analysis of the 13 cases which were fatal from paralysis shows that in them the local exudation persisted for periods varying from six to thirty-three days. The onset of the paralytic symptoms was observed on from the fourteenth day at the earliest to the forty-first at the latest, counting from the commencement of the attack of diphtheria; while the fatal event occurred at from the third to the thirty-fourth day from the commencement of the palsy.

Onset of Paralysis.		Date of death, from onset of paralysis.	
First week	0	Within one week	4
Second week.. ..	1 (14th day)	,, two weeks	3
Third week	3	,, three weeks	2
Fourth week.. ..	3	,, four weeks	2
Fifth week	4	,, five weeks	2
Sixth week	2		13
	13		

Cadet de Gassicourt in the treatise I have already cited divides cases of diphtheritic paralysis into three classes. According to this arrangement the paralysis is (1) *limited*; (2) *generalised*, or (3) of the *cardio-pulmonary* form. Cases of the first class are those in which but one or two muscles or groups of muscles are affected. De Gassicourt states that it is rare for the paralysis

to be limited to any part other than the palate and pharynx, and that this particular form of limited paralysis forms a very large proportion of all cases of paralysis; in fact he states that out of 128 cases of diphtheritic paralysis observed by himself in 103 the paralysis was so limited. My experience has not been the same as his in this respect, as will be seen on reference to the paragraph of this paper headed "extent of paralysis." Nor do I find that my cases fall readily into these three divisions. There are many cases which are intermediate as regards extent of paralysis between the limited and the generalised forms.

The large majority of cases of limited paralysis terminate in recovery. But, as in Case 10, the paralysis may fall upon an important group of muscles, those of respiration for instance, and so prove fatal.

Of the generalised form of paralysis in varying degrees, Cases 5, 6, 8, 9, 13, 15, 16 and 20 are examples. Of severe cases of recovery from generalised paralysis the following are good instances.

Case 22.—M., aged 3. Sore throat on June 7; admitted the same day with membrane on both tonsils, which had cleared off by June 18; there was a cloud of albumen in the urine; the amount of albumen increased so that on July 1 there was much; then it diminished, and after August 1 the urine was normal. On June 21 it was noted that the pulse was 108, and the heart's action at times very irregular. Next day there was a nasal voice and return of fluids through the nose. The condition remained much the same for some days, but during the first week in July the legs became affected and by the 8th the child was unable to stand, though he could move his legs in bed; the knee-jerks were lost. On July 17 paraplegia was complete; on the 18th there was inability to swallow and the heart was irregular; on the 21st the patient could not lift his head from the pillow, and on the 25th he was unable to turn himself in bed and, in fact, to help himself in any way, the muscles of the upper and lower extremities, trunk and neck, being paralysed. On July 26 the diaphragm became affected; the intercostals, however, seemed to be acting well; there was also inability to cough; on the 28th both external recti were palsied; on the 31st the pulse was 130 and regular, respiration 30; the face was continually bathed in perspiration; the diaphragm acted but slightly; the patient could only make attempts to talk; it was with difficulty that he made even a sound; one could see him make an inspiratory effort and then try to utter a syllable, each syllable requiring a fresh effort.

This state of things remained till August 5; it was then noted in addition that the saliva dribbled from his mouth. After that date, however, he began rapidly to improve; on August 10 he could swallow; by the end of the month he could sit up and during the first week in September was able to walk. He was discharged well on September 20. This is the most severe case of generalised paralysis that I have seen recover.

Case 23.—F., aged 5. Said to have first had a sore throat on April 10, but was not admitted till the 21st; there was membrane on each tonsil and a cloud of albumen in the urine, which continued up to May 29, after which date the urine was normal. There was also croup which necessitated tracheotomy on April 23; a good deal of membrane was coughed up through the tube up to the night of April 26; on April 29 the tube was finally removed; by May 29 the wound was quite healed; on this date the voice was noticed to be nasal; in the afternoon the child became collapsed and was unable to cough properly, probably because the vocal cords could not be approximated; there was pallor; pulse 102. On June 2 the pulse was 120; it had previously been 160; the child could not sit up or lift her head off the pillow. There was paresis of the left external rectus. On June 6 there were attacks of collapse and frequent coughing; the wound had broken down; (it did not heal up again thoroughly till July 17). On June 9 there was decided paralysis of the upper and lower extremities; the abdomen did not move at all in respiration, but the abdominal muscles could be felt to act when the patient tried to cough. Breathing was chiefly performed by intercostals. June 21, cough still aphonic. July 3, spoke to-day for the first time since the beginning of June in a fairly loud voice, which is slightly nasal; no squint; can swallow; paralysis of limbs better. On July 9 in the morning there was an attack of vomiting, with prostration and coldness of the extremities; at noon the pulse was 146; at 11 p.m., 90 and occasionally intermittent. July 10, pulse 100, regular. July 17, pulse 96, regular; voice still slightly nasal; otherwise the patient's condition has much improved; she can sit up but not stand. July 27, can walk by herself but is unsteady. Went home quite well on August 12.

But by far the most interesting group is the third, the cases of cardio-pulmonary paralysis (de Gassicourt), the cardiac and vagus paralysis of other writers. From what I understand, authors describe more than one class of case under the head of cardiac paralysis. Henoch, for instance, says that "risk of sudden death from failure of the heart's action hangs over the convalescent

like the sword of Damocles;"[1] and he instances cases in which somewhat sudden death took place from cardiac failure, three or four weeks after the commencement of the attack of diphtheria; there having been no other paralysis. Again Dr. Goodhart writes, "but in calling diphtheritic paralysis an affection of the convalescent, we must not forget that in the active stages of the disease we have also a paralysis which constitutes one of the gravest dangers of diphtheria—viz., paralysis of the heart; one can but suppose, indeed, that this is a part of the same tendency to the implication of the nervous centres as is seen in the stage of convalescence, and that in those terrible cases of sudden death, which are by no means uncommon both during the disease and convalescence, we have some sudden disturbance of the vagus, which thus manifests itself by means of its cardiac branches."[2] Further on in his book Henoch says: "the important connection between the nervous system and the production of this cardiac paralysis, seems to me to be indicated by the fact that these cardiac symptoms not uncommonly set in in the course of diphtheritic paralysis, even in ordinary cases of paralysis of the palate only, and cause death from syncope almost at once."

Now it seems to me that the pathology of cardiac paralysis may be different in different cases of diphtheria. First of all we have those cases in which syncope, which may prove fatal, occurs during the acute stage of the attack of diphtheria—perhaps it would be better to say during the time when exudation is still present upon the fauces or on one of the mucous membranes,—and without there being any symptom of any other paralysis (palate, ciliary, &c.). Secondly, there are the cases in which the patient having already become more or less paralysed is seized with symptoms of cardiac failure, which may and often do rapidly end fatally. Thirdly, there is the group in which the patient, being convalescent and having been free from membrane for some days or even three or four weeks, is seized with symptoms of heart-failure which also may terminate in speedy death.

Cases of the first description are so common that it is unnecessary for me to say anything further about them from the clinical point of view. Some cases of diphtheria frequently die of cardiac failure, or "cardiac paralysis," at times suddenly; at other times slowly. But the cause of this heart-failure is rather to be sought in the immediate action of toxic products of the bacillus diphtheriæ on some part of the nervous mechanism,

[1] *Loc. supra cit*, vol. ii., p. 296. [2] "Diseases of Children, 4th ed., p. 204."

than in actual changes of a degenerative nature in the nerves or muscles. Death in this manner is also seen in scarlatina maligna, and in that disease we do not hear of "paralysis." Cases 18 and 19 are examples in my opinion of this mode of death, even though in both of them paralysis of the palate had supervened; both patients were very ill before this event and were in danger of death from heart-failure quite apart from the occurrence of any other paralysis. In Case 21 also, the patient was profoundly poisoned before the onset of the paralytic symptoms. If these cases of death from cardiac failure during the presence or very soon after the disappearance of membrane are to be classed amongst cases of diphtheritic paralysis, then the numbers of paralytic cases would be much increased and the death-rate (from paralysis) be much higher. This group of cases should therefore be separated from the other two.

Of cases in which cardiac failure supervenes upon a more or less extensive paralysis, Cases 5 to 9 are examples. In four of these cases evidence of cardiac disturbance was observed during the course of the paralysis, and in three (Cases 5, 8 and 9) there was cardiac dilatation. In these cases heart-failure may have been due to disordered innervation or simply to fatty changes such as we know take place in the cardiac muscle. In those of the cases where there was dilatation these changes may well have been the cause both of dilatation and failure of the heart.

There remains to be considered the third group of cases, namely those in which death more or less sudden from cardiac failure, occurs during the stage of convalescence. I have no doubt myself that some of these cases are due to a degeneration of the cardiac muscle, and that others are due to a degeneration of the vagus or its branches. The latter cases are much less common than the former, and it is of these that I wish especially to speak.

The symptoms usually associated with an affection of the vagus are (1) alteration in the action of the heart; (2) paresis or paralysis of the intrinsic muscles of the larynx, and alteration in the rhythm of respiration; and (3) vomiting. Examples of paralysis in which these symptoms were present in varying degree will be found among the cases previously detailed, especially Cases 6, 14, 15, 16 and 23; in the last case there was recovery. In Case 16, symptoms of cardiac failure with vomiting and aphonia supervened some days after the onset of a generalised palsy, and rapidly proved fatal. But, as I have said, there was evidence in these cases of nerve degeneration, shown by the

paralysis of the limbs, &c. In the following case these symptoms (cardiac, respiratory and gastric) were present and proved fatal, quite apart from any other paralysis. The case occurred during the summer of 1892, but in consequence of the uncertainty that exists as to whether these symptoms are really due to vagus-degeneration or not, it has not been included in the statistics of paralysis given in the early portion of this paper.

Case 24.—*Severe diphtheria, faucial and nasal; cervical abscesses; (?) palsy of vagus.* M., aged 4½ years; admitted on May 10, with a severe attack of diphtheria which had commenced on May 8; there was membrane on both tonsils, a muco-purulent discharge from the nose and much cervical glandular enlargement. The patient was restless but took nourishment well; pulse 142; temperature 102°·8 F. The urine contained albumen. By May 13 the deposit (membrane) had cleared off. The boy died on the evening of May 27, and the symptoms may be stated briefly as follows:—He gradually became weaker but never paralysed anywhere; there was albumen in the urine in variable amount; on May 25 the urine became rather scanty; the pulse remained persistently frequent, 130 to 140; there was much pallor all along, for the last three or four days extreme pallor; on May 23 the heart was slightly irregular and so also was the respiration; the patient took a dozen or so deep breaths and then there was a period of apnœa; there was also occasional vomiting during this day; slight stridor on inspiration with some recession of the six lower spaces and epigastrium; this continued more or less up to the end. On the 24th the reporter notes "the respiration is very irregular; the child appears to forget to breathe and afterwards to relieve himself by deep-drawn, laboured inspirations which are not followed by the proper amount of chest movement." Pulse 132 and very feeble. On the 24th for the first time there was wandering in mind, though the patient was quite sensible when spoken to. Temperature 96°·8 F. On the 25th the apex of the heart could be seen beating violently one inch external to the nipple line. There was vomiting during the night. The child died quietly, the symptoms remaining much the same up to the end. The cervical glands had got a good deal larger and were found *post-mortem* to contain pus tensely encysted by firm tough capsules; otherwise there was nothing amiss, to the naked eye at any rate.

Here we have a patient, who, on about the sixteenth day from the onset of a severe attack of diphtheria, and about ten days after the local exudation has disappeared, is attacked with (1)

cardiac disturbance; (2) respiratory disturbance; and (3) vomiting; with which symptoms he dies in five days, there being no other definite paralysis.

There is no *a priori* reason why one or both vagi should not be the first nerves to undergo degenerative changes in any particular instance; and it is easy to understand that if such important nerves are early affected, death may ensue before sufficient time has elapsed to afford clinical evidence of degeneration of other nerves. Such cases as the one just related are, however, very uncommon.

CASE OF CEREBRAL TUMOUR IN WHICH THE INITIAL SYMPTOMS WERE CHIEFLY SENSORY IN THE ARM AND FACE. SUDDEN AGGRAVATION WITH LOSS OF HEARING IN CORRESPONDING EAR.

Post-Mortem.—Tumour found in White Substance chiefly involving Posterior Part of Internal Capsule.

BY SAMUEL WEST, M.D., F.R.C.P.

Assistant Physician, &c., to St. Bartholomew's Hospital; Senior Physician to Royal Fever Hospital, &c.

Percy B., aged 16, was admitted into John ward on January 3, 1895. He had been in good health until six months ago when he had a blow on the side of the head with a pillow. He immediately felt dizzy, could not speak plainly, and had a feeling of numbness in the fingers of the left hand, a peculiar feeling in his face, and he said he could not laugh properly. He had no pain, did not vomit, and slept well afterwards. Ever since then he has had pain in the head, has felt weak in the left hand, has been liable to drop things, and has had numbness in the finger and palm. Occasionally a sudden pain would shoot down the arm, causing it to jerk irregularly; more rarely down the left leg, causing it to give way. He could walk and run well, could stand with his eyes shut, was not giddy, and was quite able to do his work until admission.

About three months ago his sight began to get a little weak, but it was not till a month ago that it became misty. Headache he had all his life long, but it had been worse since the blow, and has been felt recently in the frontal region, especially on the right side, and has been aggravated by cough or exertion. Five months ago he began to be troubled with vomiting, which has recurred on and off ever since.

On examination the lad looked well, and there was really nothing to note, except that there was slight weakness in the left hand, but very little in the grasp, and that the sensation over the left arm in the hand and palm was distinctly impaired, but only very little. On examining the eyes there was found to be well marked double optic neuritis. Both discs were enormously swollen, but the swelling was in excess of effusion, for the vessels were tolerably distinct all over, and there were no patches on the retina. It was the condition which is usually described as "swollen discs." It was thought that the sensation in the left leg, and also in the left side of the face, was slightly impaired too, but it was very indefinite. The epigastric, abdominal and cremasteric reflexes were all weak on the left side but active on the right. The knee-jerk was absent on both sides.

What brought him into the hospital was his loss of sight and the peculiar sensations in his arm. He did not seem seriously ill on admission.

The diagnosis was made of cerebral tumour, possibly, it was thought, of a tubercular nature. A few days later he became very much worse, had more headache, vomited several times, and was more drowsy and apathetic. Some weakness was observed in the leg, and the sensation was distinctly impaired there, while the weakness of the left hand and the loss of sensation were more marked. At the same time the movements of the left side of the face were distinctly impaired, and the sensation also was obviously defective. The pupils, which had been equal throughout, reacted now sluggishly to light as well as to accommodation, but remained equal. On January 17 the patient was very much more drowsy and had passed water under him. The hearing was noted now to be defective. He could only hear a watch one inch away with the left ear, while the hearing on the right side was fairly acute. The other symptoms were much the same though slightly aggravated.

The diagnosis having been made of cerebral tumour, and the father, when he was informed that no surgical operation could be performed, nor any special treatment recommended, decided to take the patient home, where two days later he died.

A *post-mortem* examination was made.

The interest of the case lay in the diagnosis of the seat of the new growth. From the predominance of sensory symptoms from the first, which were more or less *hemiplegic in type*, it seemed that the tumour must exist in the white substance of the brain, and probably in such a position as to interfere with the posterior

portion of the internal capsule. In this connection it was very interesting to note the loss of hearing which developed towards the end of the case at the same time as the *hemiplegic* symptoms became more marked. On opening the skull the right side of the brain was noticed to be more prominent than the left, but no tumour was visible on the surface. On section, a tumour was found as large as a walnut, lying in the white matter to the outer side of the lenticular nucleus.

The bulk of the tumour was above the level of the *basal ganglia*. The lower and posterior part was surrounded by an area of softening which involved the whole of the external, and the hind extremity of the internal capsule, and extended backwards into the occipital lobe and downwards somewhat into the temporo-sphenoidal lobe. There was some small hæmorrhage in and around the tumour, the whole brain on that side being softer and more watery than normal. The tumour proved to be a *glio-sarcoma*.

Its seat fairly well explained the symptoms observed during life. The vision we might have expected to be different on the two sides, but the fields of vision were carefully examined during life and were only found to be symmetrically reduced. I suppose that the tumour must have existed for some time, and that the recent symptoms must have been due to the development of the softening, which proved to be so extensive on *post-mortem* examination.

A CASE OF UNILATERAL COMPLETE OPHTHALMOPLEGIA.

BY FRED. TRESILIAN, M.D., M.S.

THOMAS H., aged 43. When I first saw him (April 3) he complained of general malaise, and said he felt as if he had a feverish cold. During its progress, however, he developed a convergent squint in the right eye, and examination showed complete paralysis of the external rectus, the eye making no movement whatever outwards. He then had obstinate pain in the right side of the head, especially over the temple, and around the eye, which was especially severe at night, and prevented him from getting any sleep, except an occasional nap during the day. He also had frequently sudden and unexpected vomiting not occurring after meals, but when it did, any food present in the stomach was rejected. After a few days ptosis appeared, which soon became complete, and very rapidly all the ocular muscles were paralysed. The eye was now perfectly immovable, and directed straight forwards, and he could not raise the lid. There was slight proptosis, and the pupil was more contracted than the other, and acted very feebly to light. Vision, however, was very fair, but much worse than in the other eye. An ophthalmoscopic examination, which had to be done with a candle, showed only indistinctness of the margins of the disc. He had a chancre followed by a sore throat, and a skin rash, eight years previously.

There was a large sore on the scalp, over the posterior superior angle of the parietal bone; this discharged pus, and was connected by an opening with the cranial bone. There were also two small discharging sores on the sole of the left foot. These three must have been gummata. He complained of a feeling of deadness in the skin over the eyebrow, and I found there was anæsthesia, though not on the nose, nor below the orbit. Knee-jerks normal.

Two days afterwards. Pupil did not act to either light or

accommodation, and its size was $3\frac{1}{2}$ m. About two months later there was again an internal squint in the affected eye, and there was some power of movement upwards and downwards. Some condition of episcleritis was present. I found the optic disc normal. Pupil reflex still lost, accommodation also. V. O. S. normal, O.D. counting fingers. The sores on the head and foot were quite healed. The ptosis was not quite so much. In September there was still some internal squint, the ptosis had disappeared, the pupil acted to both light and accommodation. The vision was much better. After this I lost sight of him. My diagnosis was a gumma at the back of the orbit, about the region of the sphenoidal fissure, and implicating the various ocular nerves as they entered the orbit. The treatment was pot. iod. gr. 20, t.d s. The gummata were dressed with iodoform ointment.

ALCOHOLIC PERIPHERAL NEURITIS IN ADVANCED AGE.

BY ARTHUR MAUDE.

Mr. C., a robust country gentleman of sporting outdoor habits had taken stimulants to excess for certainly twenty years. There was good evidence that some eighteen years ago no unusual daily allowance was half a gallon of beer, a bottle of sherry and perhaps eight or ten liqueur glasses of neat whiskey. In spite of ample means his favourite drink was beer, and he would in his later years sit drinking beer all day, beginning at breakfast. During the summer of 1894, when over 75 years old, he would often consume two quarts of beer, a bottle of sherry and half a bottle of whiskey in one day. This large amount of alcohol never seemed to disturb his digestion or very greatly affect his appetite; and many years ago, his relatives assert, he would sit down to dinner apparently quite drunk, eat a good meal and get up "quite sober." He never had any serious illness, except broken bones from riding accidents. He "carried his liquor" in such a remarkable way that in 1892 he fell downstairs one night going to bed, but contrived to undress himself and get to bed without discovering (as his valet did next morning) that he had broken his humerus. Excellent union resulted with no impairment of the shoulder joint (under the writer's care).

For several years he had presented increasing hebetude of mind, but not to an undue degree for his age, while his memory remained excellent when roused. Even after such a severe accident he showed no signs of delirium tremens. During the summer of 1894 his general muscular power became greatly diminished, so that he could only just stand and walk with assistance. In October, 1894, he began to complain of darting pain of a severe character over the distribution of the left posterior tibial nerve. About the middle of November the hands and feet began to swell rather suddenly, while a few weeks before there were no signs of œdema.

December 7.—The hands are much swollen, the backs being quite tense, and shiny, the skin exceedingly thin and glossy; there is no swelling of the fingers and he can remove his rings easily. The skin of both hands is much discoloured and livid, with small, defined ecchymoses dotted over them.

The insteps of both feet present the same condition. There is œdema of calves and thighs, while the muscles are much shrunken. The face is puffy and bloated, but there is no œdema of the eyelids.

There has been some tendency to spontaneous ecchymoses for several years. He can just stand with assistance and shuffle a few steps with feet wide apart. There is, however, no definite extensor paresis. Tendon reflexes (patellar) quite absent on both sides (tested by Buzzard's method). Pupils contracted to pin points, no reaction to light or accommodation.

Urine 1,020, highly acid, dark from bile pigments, no albumen, no glycosuria.

Heart—feeble in action, regular, no sign of dilatation. Arteries soft for his age. Great dyspnœa on the least exertion.

No gastric or intestinal disturbance; liver not apparently enlarged.

No hyperæsthesiæ; no pain except in the left leg and thighs. No deafness.

Can move his fingers naturally and oppose his thumbs and little fingers properly.

December 10.—Both radial pulses were of fair volume, though feeble in action.

December 11.—Both pulses were found almost impalpable, though the carotid pulse was good, and the heart sounds were not more feeble.

December 12.—The fingers of the right hand, especially the two middle ones were quite blue and livid, but not swollen. The œdema elsewhere is distinctly less, probably owing to diuretics and digitalis with strychnine that he has been taking.

December 13.—Died quite suddenly after a few moments' dyspnœa.

No *post-mortem* allowed.

Remarks.—The peculiar nature of the œdema and the symptoms of local asphyxia leave no room for doubt that the condition was one of peripheral neuritis. If so the case is worthy of record from the extreme age of the patient, as it is very rarely that a man with such habits survives long

enough to manifest such symptoms at 75. The continued habit of beer drinking bears out Dr. Ross's observation on the point ("Peripheral Neuritis," p. 150). The intensity of the vasomotor symptoms as compared with the motor disturbance is striking. Contracted fixed pupils have been noted by Fischer (Ross, "Peripheral Neuritis," p. 155).

The trophic phenomena were well marked, viz., the thin, smooth, glossy skin, and dry, brittle nails, but they were probably exaggerated by old age. About a year before death there had been an irruption of "papular eczema" limited to the extensor surfaces of both arms and the nape of the neck and interscapular regions (Ross, p. 158).

The purpuric spots are also a condition found in old age; it is noticeable that they occurred more frequently and were of larger area in the right hand, possibly because the nerve trunks of this arm had been damaged by the fracture two years before.

The absence of all distinct mental change is remarkable, but it must be remembered that this gentleman had always been in easy circumstances and for years had led the life of an absolute recluse, scarcely seeing anyone except his own servants, and for several years had never left his house and gardens.

ON IMPERATIVE IDEAS.[1]

Being a Discussion on Dr. Hack Tuke's Paper (BRAIN, 1894).

I.—DR. HUGHLINGS-JACKSON.

ALTHOUGH I had not the pleasure of hearing Dr. Hack Tuke's very important paper I have read it and beg to offer some comments upon it. After its perusal I find that I have several times seen cases in which there were what Dr. Tuke calls "imperative ideas." Unfortunately I paid but little attention to these cases in a scientific regard and certainly did not view them in the wide aspect Dr. Tuke so ably presents them. From his researches on this curious and important matter I feel sure neurologists will profit greatly. One thing which impresses me strongly is that Dr. Tuke deals with the cases of morbid ideation, he mentions as they are departures from normal mental states or at least as they are exaggerations and persistencies of mental states which at their origin it would be pedantic to call abnormal.

In the Leeds address (*British Medical Journal*, vol. i., 1889) to which Dr. Tuke refers I have suggested that certain very absurd and persisting delusions are owing to fixation of grotesque fancies of dreams in cases where a morbid change in the brain happens suddenly, or when one increases suddenly, during sleep. I will not apply this hypothesis without qualification to such cases as Dr. Tuke relates. It may, however, be that certain obtrusive thoughts which otherwise might be transitory become fixed, become "imperative ideas," consequent on some very slight morbid change in the brain occurring during sleep. I suppose

[1] Since these articles were received by the Editor, we have had to deplore the untimely death of Dr. Hack Tuke, who was intending to sum up the discussion in replying to some of the criticisms.

indeed that Dr. Tuke means that a normal mental state, or at least one which is very common in persons taken to be quite healthy, becomes more or less fixed when general health is lowered or when some quasi-trifling morbid change occurs in the brain itself. Dr. Tuke writes under the head of "Dread of Dirt": "Should the general mental power be lowered, or a hereditary predisposition exist, this tendency to attend to the state of the skin becomes a passion, and results in the needless and wearisome washings of the body or the dress. Indeed it too often happens that the patient no longer perceives the folly of these ablutions, but labours under an actual delusion." We certainly have to account for the existence of these quasi-parasitical states in cases where the general mental power is but little lessened. The following remarks Dr. Tuke makes are to this point and illustrate what I said on his regarding "imperative ideas" as departures from normal or nearly normal mental states: (I italicise some words) "What strikes one forcibly in regard to most, if not all, imperative ideas is that between them and ordinary ideas *the difference is one of degree*, and that it is a most difficult thing to determine when the boundary line has been passed. This on the psychological side. And on the physical side, what we want above all things to know is what cerebral change, degeneration, or dissolution has occurred by which *the man who to-day is conscious of and smiles at an imperative idea, gravely believes it to-morrow and labours under a systematised delusion.*" Further on, Dr. Tuke writes: "The predominating idea has, in fact, become part of his [the patient's] own being." It is then a quasi-parasitical mental state of the patient, as indeed Dr. Tuke's words "predominating idea" mean.

In the Leeds address already referred to I have spoken of "fixation of states" in suddenly occurring lesions in cases which are not insanities: aphasia for one example.

I think Dr. Tuke has done good service in drawing attention not only to the long past researches of Laycock, but to important principles more recently stated by Anstie and Thompson Dickson and by Monro. Monro, so far as I know, first enunciated the doctrine that in cases of insanity the abnormal mental condition is double, that in it are two opposite elements, one negative and one positive. [Of course there is the obvious exception of complete dementia (deepest dissolution) where the mental condition is entirely negative.] For my part I consider that illusions, delusions and all other positive mental symptoms in insanities signify activities of healthy nervous arrangements of the highest cerebral

centres (so-called "organ of mind"). What we call an insane man's illusions are his perceptions, what we call his delusions are his beliefs, and more generally his positive mental symptoms sample the mentation remaining possible to him, a mentation occurring during activities of what is left intact of his highest centres, of what disease has spared. The physical condition for these positive mental symptoms is not caused, using the word cause in its strict scientific sense, by disease, not caused, that is, by a *pathological process*. Disease is, I submit, answerable only for the co-existing negative mental element of insanity. Here let me remark that, to take one kind of mental symptom, an illusion, a positive mental state implies a co-existing negative mental state; if a man sees a black cat where there is only a black felt hat, not only *is there for him* a black cat, but *this is not for him* a felt hat. Similarly, *mutatis mutandis*, for other positive mental symptoms sampling the positive element of a patient's insanity. As to the physical. Disease of the highest range of the highest centres producing loss of its function or destroying it, answers to the negative mental element in a case of insanity; whilst the activities of the lower, intact, ranges answer to the positive mental element. But sometimes the positive symptoms are super-positive as in post-epileptic unconsciousness (negative element) with mania (positive element). In these cases the lower ranges are in over-activity, and yet I think that these ranges are healthy, that they are untouched by the pathological process which has produced loss of function of, or has destroyed, the highest range. Their over-activity is an hyper-*physiological* state and is analogous to the heart's beating more frequently after section of the vagi. Here comes the application of Anstie's principle. The loss of function or destruction of the highest range of the highest centres is *not only a loss* (answering to the negative mental element of an insanity), but is also a taking off of control from the lower ranges; the "taking off" of the higher is a "letting go" of the lower. The disease may be said to be "constructively accountable" for the over-activity of the lower ranges, for, had not disease destroyed the highest range, that highest range would have continued to control the lower ranges.

I have suggested that the *rate* with which control is removed is an important factor in insanities—that the lower ranges are, at least temporarily, in greater activity the more rapidly the highest range is rendered functionless or destroyed. When disease of the highest range progresses very slowly there may be no obtrusive positive mental symptoms (control slowly removed); when it is

very rapid the patient's mentation (the mentation remaining possible to him) is at a great rate (control rapidly removed).[1]

With regard to what Dr. Tuke says in his first paragraph I submit that the scientific study of insanities may be best begun in general hospitals. For in them we encounter cases in which there are very slight departures from the patient's normal mental state; the slighter departures are most easily analysed. I have tried to illustrate this ("Neurological Fragments," *British Medical Journal*, March 5, 1892). We see in these hospitals, however, all degrees of departure from normal states from slightest temporary confusion of thought to deep coma. We should study cases of departure from normal mental state regardless whether or not they are cases of insanity ordinarily so-called, delirium in acute non-cerebral diseases, pneumonia for one example. We should, I think, find in the degrees of insanities so occurring some index of the general condition of the patients and thus some guide to treatment of their cases. I think we should learn something of great value from the analytical investigation of the abnormal mental condition in certain cases of poisoning by cannabis indica and belladonna. Dr. H. C. Wood in his work "On Therapeutics" records his experiences after taking a large dose of cannabis americana. A careful study of the abnormal mental condition this distinguished physician records will, I submit, in a scientific regard, be more profitable than that of most cases of insanity ordinarily so-called. Among other things there was "the sense of prolongation of time," a subject of great psychological interest; this "prolongation of time" occurs in dreams and in some cases of ordinary delirium.

In some cases of intracranial tumour there is a general lowering of mind or synonymously of consciousness towards and sometimes close upon complete dementia. I fear, however, we do not give enough attention to mental failure in these cases when it is slight. The degree of mental degradation will, I think, be helpful in gauging the patient's condition, especially with regard to the degree of raised intracranial pressure from the tumour. Here is a most important practical matter and one of pressing interest now that Victor Horsley has, so to speak, put the question of raised intracranial pressure on a new and definite basis with regard to several important symptoms of brain disease. I dare say it might be said of such cases where there was only much hebetude or great obtuseness that there were no mental symp-

[1] I do not go into detail here as I have recently (June, 1894) published a paper in the *Medical Press and Circular* on "The Factors of Insanities."

toms. This would be rather careless since hebetude and obtuseness are in this application words synonymous with defect of consciousness (negative mental state). In these cases the negative mental condition is most or solely marked; positive mental *symptoms* are rare, and I suppose this happens because intracranial pressure is slowly raised, the brain slowly squeezed; hence the functionable remainder of the brain will be in but little or in no over-activity; control being slowly removed.

2.—Dr. G. H. Savage.

After hearing the full and very suggestive paper read by Dr. Hack Tuke I feel that what I have to say must be rather in the way of complement to what has been so well said, while I also give the more special experience which I have had during the past twenty years. First of all, the term "imperative" ideas in one respect is misleading, for though these ideas are at times compelling they are not always the source or spring of action; they command in a way, but to command rather infers that they expect some result from the command; whereas there are certain patients who have had dominant ideas for years who have never felt that these had any relation to their actions or their conduct. Some imperative ideas are suggestive and others compelling.

As to their causation in my experience the most common predisposing cause has been hereditary predisposition to some kind, some neurosis or a tendency to nervous degeneration present in the patient's parents or other marked evidence of nervous instability in brothers, sisters or cousins.

As to exciting causes, or if I may use the term, the determinant of the form of imperative idea, I have met with many cases in which it appeared to be a steady morbid growth which had been for long under the surface, but through some accidental cause had been brought into sight. Shock may produce the effect. It may be one sudden shock, a repetition of shocks, or the result of a prolonged state of expectancy and anxiety; thus a person who has been in imminent danger for some time may become so affected that the dread is never overcome; allied to this I have met with instances where a dream of unusually vivid character has been enough to start the whole train of morbid ideas and has fixed them for ever. There is no doubt that the

nervous system under certain conditions is more prone to take these impressions and to retain them. It seems as if through certain surrounding conditions an impression is made which reaches the more fixed and automatic part of the nervous system so that it is no longer merely stored for use when it is called for by consciousness, but it appears as a reflex act, one impression bringing it up without any will being exercised. I believe that these imperative ideas are very common and that nearly everyone has some. One man cannot dissociate from his mind some notions which have no apparent relationship; these may, very probably, have arisen in the pre-conscious stage or in the stage of development before memory became established. I have the feeling, which is common I believe, about walking along a pavement. I have an inclination to avoid the cracks and at the same time I have a tendency, I own, not irresistible, to touch the iron railings with my stick when I walk along a street, but more still, I hardly ever go to sleep without just before becoming unconscious having one sentence which has relation to nothing in my past or present, but which forces itself on my mind, then I know I am going off to sleep and if for a time I have been sleepless and this sentence comes to me I know sleep is not far off.

To compare mind and body we know that what is one man's food is another man's poison, that a very trivial thing apparently will make all the difference, thus the comfort of one depends on his not taking tea, while another cannot take strawberries. The body has its imperative ideas, and I shall have to refer to the imperative ideas of the senses or rather the imperative sensations—one person being unable to stand the presence of a cat, while another is affected by a rose. The senses, in fact, give us valuable aid in considering this question, for not only can we see that these imperative nervous influences may be primary, as with the cat smell, but they may be secondary or associated; thus in the so-called photisms certain persons have associations with a sensation, which have no real relationship to them. For example, a man hears a vowel sound and sees a certain colour at the same time, whereas another perceiving a certain smell also sees a certain colour arise with the olfactory sensation. These sense relations are very imperative and are so early established that most of those who have them cannot go back to any moment when they were free from them.

In most books on the subject of recent date it will be seen that there is shown to be a very near relationship between these imperative ideas and the neurasthenic condition, also that neuras-

thenia depends to a great extent, if not entirely, on faulty association of ideas. The process has thus been described: "In ordinary course of ideation acting along ordinary lines of association, one or more ideas insert themselves and remain in consciousness with morbid intensity and persistence." Such ideas differ from delusions, hallucinations and illusions from the fact that the patient recognises that they are false. Patients may have hallucinations and illusions and not believe them, but these have a different relationship to the mind from the persistent sensations of the patient with the imperative disorder, the hallucinations are less regular in their recurrence and are not as a rule started directly by some external sensory impression. The imperative ideas vary in degree. In certain conditions a word may always insert itself, or a word may seem to escape when wanted, leaving a miserable feeling of a void which prevents rest; this is in one way like the insanity of *doubt*, to which we must refer later. Beside the simple insertion or escape of words, the word may bring up always an unpleasant association: thus the idea of dirt, of blood, of impurity, moral or religious, may arise, or certain melodies may spring up and recur without any recognised excitant. There are other allied forms of disorder in which there is not only the morbid association, but also a tendency to act on the idea. They are what a patient in Bethlem, who was very impulsive and dangerous, called a "suggestive consciousness." An idea came into his mind and had to be acted on at once. In his case there was an uncontrolled mental reflex; idea suggested action without any regard to consequences. Other examples are those who are affected by heights, by rapidly passing trains and the like. I believe nearly all such cases belong to highly neurotic families. I have been struck with the extreme horror of heights and movements which is present in those whom I have known to be neurotic. Some of these persons suffer from the dread that they shall act upon their ideas; thus one will dread a razor or a rope, while another will avoid children, lest she should push them over a cliff. In other cases we get more organised notions, and terms have been coined to express them; thus the persons who dread solitude and those who avoid multitudes have been described as suffering from claustrophobia and agoraphobia respectively. I have recently seen a clergyman who for years has been obliged to get his wife to walk about a yard behind him, because he had an unconquerable feeling that someone would jump upon his back. Doubtless there are very many other special forms which hardly deserve to have names, so long as their origin is recognised. The most

definitely and elaborately organised state of this kind is seen in many cases suffering from the so-called paranoia.

The imperative ideas may arise chiefly with the senses. I have seen several cases in which the idea that there were pins or needles in the clothes rendered the patient's life miserable. In one case a young lady with neurotic heredity asked over and over again whether it were better for her to put on her clothes, still having doubt as to the presence of pins, without examining them, or if she had better spend half-an-hour searching in vain for the pins, and then still not satisfied, put on her clothes. I advised the putting them on without consideration, and whether this or other treatment had the effect I cannot say, but any way she recovered completely. The persistent feeling of dirt or of communicating contagion are allied states to those just considered: they differ only in the explanation. In one case a middle-aged maiden lady spent all her time in dusting her home treasures, still being wretched at their supposed state of dust. In other cases a feeling that the contact with the door handle may either give or communicate poison is not very uncommon; in many of these cases there is some sexual vice, or at least some sexual abnormality. One spinster was miserable for hours after she had passed water or touched the chamber utensil; she was in the habit of putting on gloves to prevent harm arising. In another case the patient used to wear gloves lest her hands should touch the genitalia. Other notions equally imperative and equally unreasonable abound. In one case a young man otherwise healthy but of neurotic heredity was haunted by the fear that fire was lurking in all sorts of places; he thought when in my consulting room that fire might exist among the books on the table and asked to be allowed to see. He opened the pages of the various books and for the time was at peace only once more to wish to see that he had not by friction set up fire in the books he touched.

I have already said that some of the dominating ideas arise from habit, and I suppose all of us have experienced the inability to be satisfied that the front door was locked or the gas turned off. In some cases a hallucination may become an imperative idea, and in one very interesting case the hallucination had formed what I may call a mental scar. A woman had an attack of puerperal melancholia, and in this she saw her brother's throat bleeding and cut, and she was convinced that her brother had cut his throat in grief at her madness. No evidence could satisfy her that her brother was well. After months of illness she got quite well, but this hallucination reappeared

as a kind of warning when she from nursing or the like was overwrought. There seemed to be a denudation exposing the established scar. Akin to these imperative ideas connected with sensation one must not forget certain hypochondriacal cases in whom there is but one set of morbid ideas, which, however, dominate and destroy the usefulness of life. There are several medical men in London who have such imperative ideas; they believe they are dying, that their brains are unlike those of other human beings or that their general nervous system is one apart. No argument will satisfy these persons. Therefore, in a certain way they differ from very many who will admit that their ideas are unreasonable though they are uncontrollable. As a rule it is more common to meet with imperative association of ideas; these may be linked in many ways, but one idea brings up as a certainty the corresponding one.

Years ago I had under my care a man who, having lived in India for some years, was once with others in great danger from a rising of the natives. It was hourly expected that their fort would be stormed, that the men would be killed and the women outraged. So it was agreed that the men should, if occasion arose, kill their womenkind and then fight to the death. They were relieved, but the association between wife and killing was so firmly established that the patient felt his only safety rested in his living in an asylum. This he did till his wife died; after that he went out and lived at liberty.

Volition is affected in several ways, but the chief disorder is shown in some of the cases of *doubt*, in which the patient is never able to make up his mind as to what to do. In some there is the doubt about movement. I have letters in which patients ask over and over again if there is any harm in their going more and more slowly, if by so doing they can satisfy themselves.

I have known a young man of this type who nearly lost his life as a result of not being able to make up his mind; in the particular instance it was a question whether he should shoot at a bear, and if a companion had not, his future chance would have been done for.

Among the associated ideas which are imperative some are strongly allied to the emotional side. One has met patients who seemed to have had their emotions perverted so that they invariably laughed when they ought to have shown sympathy. In some cases the sexual attraction which may be either normal or abnormal may be persistent, recurring and dominant, and there are certain associations of this kind specially noteworthy. Thus

certain associations produce ideas of sexual impotence, or some very slight things may during the earlier days of marriage set up imperative ideas which produce loathing, hatred and impotence, which will be dangerous to one or both of the parties. A young married man finds from certain acts that his wife is not a virgin, or he may simply get the notion into his head and it may dominate his life.

Similarly the judgment may be in abeyance as in the "*folie du doute*." I do not for a moment intend to assume that the parts of mind are distinct entities, but it is well to note that certain faculties are more affected in one case than in another. In some cases there is a morbidly scientific spirit. One patient who was under me in Bethlem could not resist the temptation to see whatever was written if he could, so that in a train or an omnibus, in a house or in a dining room, if a man opened a letter he could not help trying to read over the owner's shoulder. He was struck by the owners of the letters several times, but still he felt the overmastering desire to know. He is *Grubelsuchtic*. He has several times been in danger of his life when, seeing a piece of paper revolving round a cab wheel, he has tried to get it. He has gone home miserable after failing, and has returned to the street at night to see if he could find it. This man has been for the last twelve years in this state and from time to time comes to see me, for I have been anxious to watch his course. He is no more weak-minded than he was and manages to earn a living, though his peculiarity interferes with his doing full work. As to the curability of these imperative ideas, I can only say that if they have existed for a year or more I do not think there is any prospect of cure. If they are acute and if associated with any special cause, or if associated with a period of life, such as adolescence or the menopause, there is some slight hope. Few of these cases need to be permanent inhabitants of asylums, in fact, many of them need never be there if their friends can afford to keep them out of asylums.

Then, to conclude, in my experience there occur certain organic nerve defects associated with abnormal mental reaction which may show themselves on the organic or visceral side, on the sensory side, either simply or by association; on the voluntary side with morbid impulse, and on the intellectual side by a further development of the association of ideas, these latter being more commonly called imperative ideas, though they do not differ from the sensory or visceral disorders.

3.—Dr. C. Mercier.

The feature in Dr. Tuke's paper which impresses me most, is the complete acceptance of a doctrine that I have long held and preached, but which has never before, as far as I know, had any adherent, that insanity and disorder of mind are not convertible terms, and that disorder of mind—grave and prominent disorder of mind—may exist in a sane person, and without the question of insanity being even raised. Another favourite doctrine of mine, on which I am fully in accord with Dr. Tuke, is the importance of noting the earliest departures from the normal if we would discover the true nature and origin of mental disorder. It is in this connection that the observations of Dr. Wilks are so interesting. That the trifling forms of obsession to which we are all liable—the persistent and often annoying recurrence in memory of a word, a line of poetry, or a few bars of an air—are of the same nature as the graver imperative ideas of which Dr. Tuke has given us so many instances, there can, I think, be no doubt, and hence it becomes important to note the conditions under which these trifling forms occur.

Speaking from my own experience, they do not occur when the mind is fully occupied and the attention engrossed in some intellectual pursuit—in reading, writing, calculating, playing chess or whist. Nor do they occur when the body is inactive and the mind is pursuing some train of thought—say on a railway journey—nor when bodily activity is accompanied by close exercise of attention, as in games of billiards, bowls, &c. The conditions under which they occur, and under which they seldom fail to occur, are when we are undertaking some bodily labour of a more or less monotonous character, which demands considerable muscular activity, which does not require close or unremitting attention, but which calls for so much and such frequent exercise of attention as prevents us from carrying on any continuous and connected train of thought apart from that devoted to what we are occupied about. It is, in short, when we are engaged in some handicraft, such as carpentering, joinery, smith's or other metal work, gardening, and such like occupations, that these strains of reminiscence arise and haunt us, and become, for the time being, imperative ideas. If, then, we can identify the psychological condition that obtains during our occupation in these handicrafts, we can get some insight into that which permits of the occurrence of obsession in its graver forms.

In the graver cases of the malady we must suppose that a

local portion of the brain has attained a habit of spontaneous activity which overpowers the control of those higher regions to which by its anatomical position it should be subject—that at length the whole concentrated and focussed energy of the higher nerve regions, the local direction of which is the physiological basis of will, is insufficient to overpower the rebellious activity of this revolting section; whether the imperative idea remains an idea, or whether it finds expression in word or act, is purely a matter of degree, and affects in no wise the explanation of the physiological process which is the basis of both idea and act.

The psychological condition in the slighter forms of which mention has been made appears to be this: there is a considerable degree of alertness of attention, and at the same time the actual pabulum that is supplied to the attention is insufficient to keep it fully occupied. The attention is concentrated upon the work in hand, not continuously, but at intervals only; and in the intervals, during which it is not so occupied, but is still alert, it has presented to it pabulum from the field of sub-consciousness, about which it becomes occupied; and the states which should normally be subconscious are thus lifted into the light of full consciousness, and become subjects of attention without direct guidance from the will, which is otherwise engaged. A state which has once thus arisen and presented itself to the attention in an unoccupied interval, tends, so long as the work is approximately the same, to repeat itself in obedience to the law of association, and thus it happens that the image, the musical air, the form of words, which has once arisen in one of these unoccupied intervals of attention, tends to occur again and again as these intervals are repeated. Briefly, the psychological condition may be stated as the capture of the attention during the abeyance of the will. If we take this expression and apply it to the more serious of the cases that have been brought forward, we shall find that it applies to them also. The imperative idea is in every case one that forces itself—if I may be allowed for a moment to speak metaphysically—upon the attention, and that cannot be, or at any rate is not, excluded by the will. The will is, so far as that subject of attention is concerned, in abeyance.

The physiological process which underlies this psychological condition appears to be this: returning now to the trifling and quasi normal instances of the malady, those instances in which an idea repeats itself with undue frequency and force during the execution of some handicraft operation. The operations of the

handicraft necessitate and imply great activity of the middle centres. At the same time there is very little call for the activity of the higher centres. No general, or intense, or widespread activity of the centres which are the substrata of elaborate, intellectual operations, and especially of verbal operations, is required, nor is there in the work in hand any outlet for such activity if it is evoked. Yet the high activity of the middle centres cannot go on without spreading, to some extent, in an upward direction, and starting activity in the more unstable and more easily discharged centres above them. It cannot, at least, fail to evoke some activity of these higher centres in those persons in whom activity of these higher centres is habitually great, and their tendency to have this activity easily evoked is consequently also great. During the handicraft operations, certain of these higher centres are therefore started, and in the case supposed they are centres the substrata of verbal utterance. Hence forms of words—successions of words—arise in the mind. Were these higher centres the seat of widespread activity, proceeding normally, its focus swaying hither and thither as multiform successions of verbal utterance arose, the activity started by the spread of discharge from the middle centres would in great part be drafted off to the scene of greatest activity, and what ideas accompanied the remainder would be overpowered by the more vivid and voluminous ideas thus rendered possible, would remain subconscious, and would never occupy the attention. But since these small areas, started by the upward spread of the discharge, are practically the only scenes of localised activity in the higher centres, the ideas which they represent are practically the only verbal ideas present in consciousness; and as such they seize upon the attention in every vacant interval, and are thus brought prominently to the front. That local concentration of widespread energy which is the physiological equivalent of the will, is directed upon the middle layers and goes to direct the handicraft operation. It passes by the active articulatory centres and leaves this latter unenveloped in its rushing volume, free to assert itself, to set up a somewhat independent and almost isolated centre of discharge, which has its mental equivalent in the imperative idea.

4.—Dr. J. Milne Bramwell.

At the conclusion of his interesting article on "Imperative Ideas," Dr. Hack Tuke states that the mental phenomena which he has described have been more clearly recognised by French and German, than by English writers. This is undoubtedly correct; but, although the former have long recognised such conditions, it is only within comparatively recent times that they, like ourselves, have ceased to confound them with various forms of insanity. Dr. Ladame, of Geneva, for example, in referring to the different opinions expressed in reference to "folie du doute" and "délire du toucher" by Schüle, Magnan, Krafft-Ebing, Marcé, Jules Falret, Morel, Lasègue, Ball, Meynert, Kræpelin and Scholz, says "folie du doute" is regarded by contemporary writers sometimes as a symptom of the most varied mental affections, sometimes as a psychopathic episode of hereditary degeneracy, sometimes as a special form of psychosis, and sometimes as a simple elementary psychic trouble, dependent upon the general pathology of mental alienation. Thus, doubt, he says, does not only exist among the patients, it has passed into science and could equally be called "folie du doute" on account of its uncertain place in the chart of mental maladies, as well as for the strange symptoms which characterise it.

According to Ladame, Pinel at the beginning of this century first commenced to recognise "folie du doute," while Esquirol in 1838 published the first detailed clinical observation of "délire du toucher" and laid stress on the patient's constant fight against obsessions and his recognition of their absurdity. Brierre de Boismont in 1853 also stated that the irresistible idea keenly combated by the patient was characteristic of these cases. "There is a distinction to make," he said, "namely, that ideas may make one ill when they dominate the mind, but one is not really mad except when the will has become powerless to control the impulsions." That the patients fight against their obsessions without succeeding in getting rid of them, was particularly insisted upon in the discussion upon monomanias at the Medico-Psychological Society of Paris on June 26, 1854. Delasiauve in 1859 made a first attempt to distinguish between pseudo-monomanias and recognised forms of insanity. After 1860 observations on "délire du toucher" became more numerous, and Baillarger appears to have been the first to notice that these conditions frequently commenced at puberty. Marcé in 1862, though he failed to separate "délire du toucher" from insanity, clearly

described the origin of imperative ideas. "In a predisposed person," he says, "feeble of character, endowed with keen sensibility, a word, an emotion, a fear, a desire leaves one day a profound impression. The thought, born in this manner, presents itself to the mind in an importunate way, takes possession of it, does not leave it, dominates all its conceptions; during this time the individual may have consciousness of all the absurdity, unreasonableness or criminality of this idea, the acts themselves soon conform to these unhealthy preoccupations and become absurd and extravagant." The term "folie du doute" occurred for the first time in the celebrated discussion upon "la manie raisonnante" at the Medico-Psychological Society of Paris, March 26, 1866, but from what was said by Jules Falret on that occasion it was evident that what he called "folie du doute" corresponded to "délire du toucher" and not to the form of mental trouble described two years later by Griesinger as "Grübelsucht" and "Krankhafte Fragesucht." In this discussion Baillarger stated that one of his patients never ceased to make suppositions and to lose herself in "if" and "perhaps." A clear distinction between these diseases and recognised forms of insanity was made for the first time in 1866 by Morel, and he stated that patients suffering from imperative ideas did not interpret their obsessions after the manner of the insane, that they neither experienced hallucinations nor illusions, nor underwent those transformations which change the personality of the insane and make them radically different from what they were before. Heredity, not only hereditary insanity, but also other nervous conditions such as hysteria and hypochondria he regarded as important exciting causes. In 1868 Griesinger published cases which showed for the first time that the entire malady might consist in insoluable questions, which unceasingly pursued the patient who could not escape from "why," and "how," and he considered the condition might be independent of emotional complications. In 1875 Legrand du Saulle showed that the same patients could present successively the symptoms of "folie du doute" and those of "délire du toucher," and he attempted, by joining these two conditions, to form a special mental affection having a veritable morbid entity. In 1877 Westphal published an important contribution to this subject. According to him, the obsession never becomes a true "idée fixe délirante," but always remains a stranger to the patient's ego, while the insane conform logically to the deductions of their fixed ideas. This scientific distinction between fixed ideas and obsessions has long been recognised by the Church.

which has always made a difference between possession and obsession, saying for example: "This man is not possessed, he is only suffering from obsession." According to Westphal an obsession is not an emotion, nor is it ever produced by one, and if attacks of mental agony appear later, these are always secondary and simply concomitant phenomena. This opinion is opposed to the observations of Morel and the French savants. Wille holds that obsessions can have an emotional base, and thinks that they are sometimes followed by veritable mental alienation, while Westphal affirms that cases of obsession never become insane. Wille, as well as Legrand du Saulle, has noticed the frequent suicidal ideas and even attempts at suicide, sometimes followed by death, and considers "folie du doute" and its likes as an intermediate condition between the nevroses and the psychoses, and that obsession is always ready to pass into madness.

Westphal divides obsessions into three kinds:—(1) Those which remain purely theoretical as the "folie du doute," when it takes the form of questions. (2) Those which produce certain actions as the "délire du toucher." (3) Impulsive obsessions which provoke immediate actions.

While other authors since Morel have emphasised heredity as an essential factor in obsessions, Magnan in 1885 appears to have been the first to consider this mental trouble as a direct and immediate sign of morbid heredity.

In opposition to Legrand du Saulle, Ladame demands the separation of "folie du doute" and "délire du toucher" into two distinct clinical varieties. These, like pleuro-pneumonia, are certainly often associated, he says, but more often exist isolated. In illustration of this he cites the following cases:—

(1) A young man, aged 28, suffered from fear of contamination and frequently washed his hands as a method of purification. He never feared he would forget anything and never addressed questions to himself. This, according to Ladame, was a case of "délire du toucher" without "folie du doute."

(2) Miss ——, aged 33, has since childhood had ideas which she could not get rid of. She asks herself all sorts of questions and seeks in vain for their answers. These are particularly in reference to the other world and the creation. Did everything make itself? Has God created all things? Did not the world make itself? Is there a God? How can one divide objects into infinitely little parts, when each little part can still be divided? How is it that an object infinitely divided can still be divided, notwithstanding that one cannot divide it any more? Is God able to divide it

still further? God only can divide it and nevertheless this particle cannot be divided! How do you account for that? She has never suffered from "délire du toucher."

Van Eeden says that in four cases treated by him the connection between "folie du doute" and "délire du toucher" could not be mistaken, and that in all these cases the "folie du doute" was evolved after the "délire du toucher" and was an evident consequence of it. One case commenced with the fear of catching syphilis. The patient avoided touching anything he suspected of being contaminated and, if this happened, scrupulously washed his hands. His precautions became exaggerated, until he not only doubted their efficacy, but also the clearness of his judgment, and then, despite the excessive attention given to these washings, could not obtain a certainty of complete cleanliness. A similar progressive evolution was observed, says van Eeden, in another case, where the patient suffered from "crainte de souillure," then doubt and finally mistrust of herself. The "délire du toucher" began with fear of soiling her food, she washed her hands incessantly, but afterwards the doubt returned. Another patient, who was nursing a case of cancer, became afraid of catching it. She washed her hands constantly and avoided coming in contact with suspected persons.

In agoraphobia, claustrophobia, &c., van Eeden says the idea of fright arises suddenly from the impression of the surroundings, and it is only after having this that the patient avoids open or closed spaces. Here it is the fear which causes the obsession. He classifies these and similar conditions as follows:—(1) "Conceptions obsédantes"; here the obsession springs from a precise and detailed conception of some act which acquires an impulsive force. (2) "Les émotions obsédantes"; chiefly terror, in which a momentary emotion, an impression—which a normal man could equally well experience, but which he quickly represses—dominates the volition and the reason. The patients are not able to say what they fear, or if they give a reason it is evidently an invented one. (3) "Impulsions obsédantes"; the irresistible tendency to commit strange or improper acts. (4) "Les idées obsédantes" properly so-called; the intellectual obsessions of the French, the "Grübelsucht" of Griesinger and Berger. The patient is not able to escape from the obsession to think constantly about a certain subject or question. Here it is neither a question of a perception or emotion, nor of an impulse to commit an act; there is only one isolated idea—a word, a phrase, incessantly pursues the patient and continually occupies his thoughts.

Van Eeden regards what he calls "manias of superstition" as an interesting variety of obsessions. One of his patients, a man aged 40, of healthy constitution, has since childhood attached prophetic signification to puerile facts and events. To wear a certain necktie promises him happiness or unhappiness. If he does not touch a certain boundary he thinks evil will happen to him. If he does not re-read a certain line or make a certain letter thicker in writing something horrible will befall him. At first his strange ideas were insignificant, or he was able to resist them, but as he grew older they filled his life and rendered it intolerable. For twenty years he made a pilgrimage every Sunday to the railway station in order to kick a certain post three times with each foot. If he did not do this his father would die. In order to rid himself of these obsessions he makes vows and associates threats with them. He says for example: "If I yield to one of my caprices in the course of an hour I shall have apoplexy before twenty-four hours have past." At first this succeeded, but soon the effect of the vows diminished, and he was compelled to make them stronger. The unhappy man now stands sometimes for a quarter of an hour muttering the most fearful imprecations, in order to get the strength to go an errand. If he omits them he is forced to obey the most absurd impulses. He must stop before a certain house, retrace his steps, touch boundaries, stop passers-by or touch their clothes; in a word he is obliged to act like a maniac. His intellect is perfectly normal, and he attends to his business as if nothing were the matter.

Bérillon thinks the professional character of these nervous troubles has not been sufficiently noticed, and he draws an analogy between them and the different functional spasms which show a tendency to professional localisation. In illustration of this he cites the following cases:—A young priest, not timid in the performance of his other religious duties, suffered agony on entering the pulpit. Another suffered in the same way when he received a confession. A medical student suffered extreme agony at the sight of a few drops of blood. A chemist made up a prescription which caused the death of a customer. He was able to prove that it was dispensed exactly as ordered by the doctor, but as his existence became a veritable torture from constant fear of making a mistake he sold his business. A notary only had morbid fears when he had to give a professional opinion. A hairdresser noticed that his hand trembled one day, and then constantly dreaded that this would reappear when he shaved his best customers. The same anxiety did not exist when he had to

shave a poor or unknown customer. Dr. Frémineau reports the case of an actor who abandoned his profession on account of extreme stage fright. This condition only appeared after a successful career. Dr. Bérillon reports several similar cases. Riegler has noticed a morbid fear amongst railway mechanics, to which he has given the name of "sidérodromophobie"; this is characterised by an extraordinary aversion to their habitual occupation, and the sight of a train or the whistle of an engine is sufficient to revive their anxiety. Grasset mentions that a distinguished Parisian surgeon commences to be anxious the moment a patient leaves his consulting room with a prescription. He anxiously asks himself whether he could have written centigrammes instead of milligrammes, and only recovers his mental calm when his servant, sent to seek the patient, brings back the prescription and he can see that it is all right. Another doctor, he says, is rendered perfectly miserable by the fear of microbes. Brochin reports the case of a doctor who fears no contagious malady, except diphtheria, and who shows proof of veritable heroism every time he sees a diphtheritic patient. A case has recently been reported from abroad, where a medical man, dreading that his fees might be the means of contagion, invented elaborate methods of sterilising them, and I know of a similar case in this country.

These morbid fears are awkward enough when they occur in ordinary life, but according to Bérillon they acquire graver importance when the subjects of them are liable to enforced military service, especially in the ranks. In Legrand du Saulle's classic case of agoraphobia, a lieutenant of infantry experienced undefinable agony when obliged to cross an open space in civilian clothes, but this never happened when in uniform. When an officer suffers in this way he can escape from the intolerable situation by getting leave of absence, but it is not so with the soldier. Bérillon gives amongst others the following examples:—

Paul C., aged 25, illness dates from military service. When ordered to cross the horizontal bar at the gymnasium he was seized with extreme apprehension, and afterwards the idea of this exercise was always present. Again ordered to perform it he became terrified, and asked to be excused, but from this time his superiors insisted more and more upon the dreaded exercise. One day in attempting to cross the bar he became giddy and fell upon his head. A veritable agoraphobia developed, and since his return from military service he has been unable to cross a road by himself.

M., aged 37, commercial traveller, suffered from chronic diarrhœa, which caused him to dread leaving home; after this ceased he retained his nervous fears. Once, when a short distance from his house, the anxiety was so strong that he was obliged to return, and since then, seven years ago, he has lived in a circle having a diameter of about 200 metres, and nothing will induce him to leave it. When, as a member of the Reserve, he had to serve his first period of twenty-eight days he was able to perform his duties, but at the second period the agoraphobia had developed, and by dint of diplomacy, ingenuity and bribery, he managed to be kept constantly employed in the barracks.

According to Dr. Gélineau a crowd of sentiments of repugnance, &c., which the laity group as aversions closely resemble the conditions we are discussing. Henry the Third, for example, who showed his bravery at the Siege of La Rochelle and elsewhere, could not bear the sight of a cat. The Duke of Epernon fainted at the sight of a young donkey. Ladislas, King of Poland, got frightened, and ran away when he saw apples, and Favoriti, a modern Italian poet, could not bear the smell of a rose. Dr. Pierre d'Apono was so frightened at the sight of milk and cheese that he fainted. Montaigne says: "I have seen more people driven to flight by the smell of apples than by arquebuses, others frightened at a mouse, made sick by the sight of cream, or by seeing a feather bed shaken."

Ribot applies the term "fixed ideas" to the states we are discussing, and regards them as "chronic hypertrophy of the attention"; the fixed ideas being the *absolute*, attention the *temporary* predominance of an intellectual state or group of states. The fixed idea is attention in its highest degree and marks the extreme limit of its power of inhibition. There exists, he says, both in normal attention and in fixed ideas, predominance and intensity of a state of consciousness; this is more marked however in the fixed idea, which is permanent and disposes of the important psychical factor—time. "In attention this exceptional state does not last long; consciousness reverts spontaneously to its normal condition, which is a struggle for existence between heterogeneous states. The fixed idea prevents all diffusion. There is no antagonistic state that is able to overthrow it. Effort is impossible or vain. Hence the agony of the patient who is conscious of his own impotency." The following is Ribot's conception of the probable physiological condition associated with fixed ideas:—"In its normal state the entire brain works; diffused activity is the rule. Discharges take place from one group of cells

into another, which is the objective equivalent of the perpetual alterations of consciousness. In the morbid state only a few nervous elements are active, or, at least, their state of tension is not transmitted to other groups. Whatever may be their position in the cerebral organ, they are as a matter of fact isolated; all disposable energy has been accumulated in them, and they do not communicate it to other groups; whence their supreme dominance and exaggerated activity. There is a lack of physiological equilibrium, due probably to the state of nutrition of the cerebral centres." Ribot refers to Westphal's recognition of the difference between fixed ideas and insanity, and his statement that "the fixed idea is a formal alteration of the process of ideation, but not of its content." The "formal" perturbation consists, says Ribot, in the inexorable necessity that compels the association always to follow the same path. "There is alteration, not in the nature, the quality of the idea, which is normal, but in its quantity, intensity and degree." Thus it is perfectly rational to reflect upon the usefulness of bank notes, or the origin of things, and this state differs widely from that of the beggar who thinks himself a millionaire or the man who believes himself to be a woman.

Treatment.

The following cases, from my own practice and that of others, illustrate the value of hypnotic suggestion in cases of "imperative ideas."

(1) Mr. A., aged 24, consulted me in May, 1889. Some months previously he had a number of diseased glands removed from the face and neck, and went up the Mediterranean to recruit. While crossing a plank he fell and injured his perineum; an abscess formed, which burst externally and into the urethra. When I saw him there was a large unhealthy wound, through which the urine escaped. I instructed him to pass a soft catheter regularly, and the wound became more healthy. One day he was impelled to empty his bladder before he could pass the instrument and the water again escaped from the wound. This happened more and more frequently; at last the idea of passing water at once caused him to empty his bladder, no matter where he was at the time. This appeared to be entirely independent of the physical condition of the bladder, which did not contract because it was full or uncomfortable, but because the idea of urination presented itself to the patient's mind and was instantly translated into its physical equivalent. He now thought constantly about his condition,

which became greatly aggravated in consequence. He began to sleep badly and awoke frequently during the night; the instant he did so he thought of his bladder and was immediately compelled to empty it. Despite all treatment this continued for several months, and his state became a grave one. I had not previously employed hypnotism, but the mental element in this case seemed so marked that I determined, as other treatment had failed, to try what it would do. After explaining to my patient, an educated man, that I had no practical, and only slight theoretical, knowledge of the subject, I proceeded to hypnotise him by Braid's method. In a few minutes his eyeballs rolled upwards and inwards and he became lethargic. I repeated this the two following days, and then suggested during hypnosis that he should cease to think about his bladder, should always be able to pass his catheter, retain his urine eight hours and sleep well. These suggestions were immediately fulfilled; from that day there was no return of his troublesome symptoms and the wound healed without operation in about twelve months.

(2) Mr. B., aged 25, first consulted me March, 1890. Formerly strong and athletic, distinguished football player, bicyclist, &c. Two years previously, after the death of his mother from cancer of the breast, he began to fear that he might contract the same disease. This idea grew stronger and stronger, he developed neurasthenic symptoms, and suffered from insomnia, depression, dyspepsia, &c. Finally, the dread of cancer passed into the firm conviction that his left breast was affected by it. He now remained nearly always in one room and would not pass into another without muffling himself up and putting on an overcoat. For some months he complained of difficulty in moving the left arm and carried it in a sling. I found nothing to justify his fears, but the muscles of the arm were distinctly wasted from disuse. He was easily hypnotised at the first attempt, and this was repeated nearly every day for a fortnight, when deep hypnosis with somnambulism was obtained. His morbid ideas at once disappeared and his general health speedily improved. A few days after the treatment was abandoned, I saw him driving a spirited horse and holding the reins in his left hand. A week afterwards he told me that he felt perfectly well, and was just going to jump a young horse over some hurdles. Up to the present date there has been no relapse.

(3) Mr. C., aged 30, consulted me July, 1890. Had good health up to six months ago, when he was frightened by being awakened by his sister, who had acute mania, entering his bed-

room. From this date he suffered from insomnia and dyspepsia, lost weight and strength, became melancholic, shunned society and had the fixed idea that he would become insane. Hypnotised five times from July 3 to July 7, 1890. Deep hypnosis was easily induced at the first attempt and he slept well each night. Recovered. No relapse up to May, 1891, when he died of influenza.

(4) Mr. D., aged 43, first consulted me September, 1892. His illness commenced in early childhood, with the fixed idea that everyone noticed and criticised him and he soon began to blush if looked at. This condition had continued ever since and had latterly become worse and made his life a torture to him. Not only did he blush in the most excessive manner when anyone, even his own children, looked at him, but the thought that anyone might look at him was sufficient to make him blush even when alone. He felt that he must give up business and had suicidal ideas. He suffered from various neurasthenic symptoms. I attempted to hypnotise him five times without result. He returned for treatment in April, 1893; his condition was worse and he was in despair about himself. I hypnotised him forty-two times up to October, 1893. Slight hypnosis was induced at the fifteenth attempt, and afterwards became fairly deep, when he commenced to improve and before the close of the treatment his morbid symptoms had disappeared. Recent news confirms his recovery.

(5) Mrs. E., aged 36. Educated and highly intelligent. One of her brothers is congenitally deficient in intellect, and she has suffered from fits of depression since childhood, which have become more frequent and prolonged during the last fourteen years. The mental condition of one of her children, born during one of these periods, causes grave anxiety, and a younger child stammers. Four and a half years ago, after the death of a relative who had exercised an unfriendly influence on her life, she constantly thought that various misfortunes that were happening to her would give him pleasure; this idea increased and developed until she felt that he knew everything she did and gloated over her troubles. Finally she became possessed of the idea that she was haunted by him and felt conscious of his presence. Her whole life was filled with these thoughts, and she ceased to care for society or intellectual pursuits. She was constantly depressed, slept badly, and was weak physically. I first tried to hypnotise her on November 26, 1892, and repeated the attempt a hundred times without success, until April 25, 1893; her condition mean-

while growing steadily worse. Slight hypnosis was induced on April 26, and repeated twenty-three times up to July 21, 1893. Immediately after the first induction of hypnosis she rapidly improved and the morbid ideas and depression entirely disappeared. She quickly regained strength and interest in life and up to the present date there has been no relapse.

(6) Mrs. F. I have received the following notes of this case from Dr. F., the patient's husband: "My wife has suffered from myxœdema, following influenza; she had low temperature, loss of hair, dulness of intellect, slowness of movement, general irregular swelling of the body, facial disfigurement, alteration of voice and muscular pains. I put her on thyroid extract in January, 1893, and although the symptoms peculiar to myxœdema disappeared, she became utterly sleepless, her limbs trembled after the least exertion and her digestion remained very bad. I brought her to you on March 1, 1894, to see whether suggestion would procure sleep. At the second attempt you succeeded in inducing very slight hypnosis and she began to sleep fairly well. For more than a year she had never had more than three hours' broken sleep and often far less. She soon began to sleep thoroughly well and uninterruptedly, and her digestion, for which I had found drugs and careful dieting ineffective, disappeared after a few suggestions. Her legs became stronger and her energies restored very much to what they were twenty years ago, when she was renowned amongst her acquaintances for her untiring activity. But the fact that strikes me most forcibly is this. Several members of her family are sleep-walkers. She used to walk in her sleep in childhood, and once or twice as a young woman, and the habit is transmitted to my youngest girl. When her first baby was born—sixteen years ago—the thought crossed my wife's mind: 'What if I walk in my sleep and do an injury to my child?' I endeavoured to persuade her that she had grown out of the habit, but the attempt was wholly fruitless. The idea grew until it assumed the character of an 'idée fixe' and she always tied herself to the bedpost at night. All attempts to break herself of the habit were failures, and if she went to bed without fastening herself she was never able to go to sleep until she did so. When we moved into our present house, three and a half years ago, she became alarmed at the great height of the bedroom windows from the ground and their lowness from the floor. She began to suggest that possibly she might undo her own knots during her sleep and get out of the window. I pointed out how unlikely it was that she should walk in her sleep after a score of

years complete immunity. She granted my reasoning was just, but it did not dispel her fear, and she insisted upon my tying her to the bedpost each night in a very effective manner. In May, 1894, I told you of this persistent dread and asked you to suggest that she should neither walk in her sleep, nor be apprehensive of so doing. During that *séance* you repeated this suggestion two or three times, but have not done so since. The effect was magical. She has never asked me to tie her to her bed from that day and tells me she has never once thought about it. To me it is all the more remarkable as the hypnosis in her case is so slight and appears to pass into natural sleep if you leave her for a few seconds. She is of a nervous, excitable temperament, but by no means greedy of the marvellous or ready to accord belief to any doctrine. She had a healthy scepticism of the possibility of anyone hypnotising her, but was anxious for the attempt to be made as she suffered so acutely."

I saw this patient twelve months later, when she told me she remained entirely free from her morbid fears.

(7) Mr. G., aged 28, first consulted me April, 1894. His father was very nervous and passionate and had suffered from "brain fever" and chorea. The patient is slight and undersized, and suffers from various neurasthenic symptoms. At the age of 14, he had many religious doubts and fears, and believed he had committed the unpardonable sin. At the age of 16, while working in a cocoa manufactory, he began to fear that the red lead which was used in fastening certain hot pipes, might get into the tins containing cocoa and so poison people. This was the commencement of a "folie du doute" and "délire du toucher" which have never since left him. Instead of going on with his work he was irresistibly impelled to clean and reclean the tins. The following is taken from the letter of a friend to whom he confided his troubles: "On October 1, 1891, he told me that he had attempted to commit suicide, as his life was so miserable [he had taken poison]. He had read of a case of poisoning through eating chocolate and connected himself with it, though it was five years since he had helped to manufacture any. He now believed he might have been careless with the moulds and thus have produced a poisoned chocolate, which years afterwards had caused the child's death! The grotesque absurdity of the story, as he related it to me, would have made me laugh, had I not felt how terribly real it was to him. His vivid imagination had pictured every incident of the tragedy; the child buying the chocolate, running home full of happiness, then becoming ill and gradually

sickening in awful agony till released by death. The keenness of mind with which he sought to prove the reasonableness of his belief that he had poisoned the child was extraordinary. He wrote: 'Yesterday I was unscrewing some gas burners in a provision shop and got some white lead on my hands, and I have been thinking that it may have got amongst the food.' I found that brooding over this fancy had brought him to the verge of despair and for weeks his life was a perpetual agony. He has worried himself about his work of fixing advertisement plates to walls and can never persuade himself that they are securely fastened. He fancies the nails are bad, or the mortar loose, and makes himself ill over it. I have pointed out to him that if a plate fell it would almost inevitably slide down the wall. This has not prevented him from painting a most elaborate mental picture of the decapitation of an unfortunate youngster, who happened to be playing marbles with his head against the wall. To enumerate all his troubles would take a small volume. I have a great pile of his letters before me now, and I suppose they constitute one of the most extraordinary analytical autobiographies it would be possible to find. In reading them I cannot help marvelling at the strange unshapely wonder of such an imagination. He makes every incident in his life the foundation stone of a castle of fancies, and of late years each castle has become a prison—a torture chamber in which he has dissected his motives and his actions until he has ceased to believe in himself at all."

When I first saw this patient the "folie du doute" and "délire du toucher" were constant and most varied in their manifestations. If he accidentally touched persons in the street, he began to fear that he might have injured them and exaggerated the touch into a more or less violent push. If the person touched were a woman, he feared that she might have been pregnant and that he might have injured the child. If he saw a piece of orange peel on the pavement he kicked it into the road, but soon afterwards began to think this was a more dangerous place, as anyone slipping on it might strike his head against the curb-stone, and so he was irresistibly impelled to return and put it in its former position. At one time he used to bind himself to perform certain acts, by vowing he would give God his money if he did not do them. Then sometimes he was uncertain if he had vowed or not. Owing to this he gave sums to religious objects which were quite disproportionate to his income. Apart from his peculiar fancies I found the patient perfectly rational and intelligent, and though his "délire du toucher" hindered him greatly in his work, he

generally managed to execute it, though on some occasions he was compelled to abandon the attempt. At this time I tried to hypnotise him on twenty-four occasions, but apparently without success and he was then compelled to leave town. He returned on April 2, 1895, for a week's further treatment and told me that since his former visit his morbid ideas had not been so frequent and marked and were accompanied with less mental agony. His condition, however, leaves much to be desired.

(8) Mr. H., aged 33, tall, strong and athletic, first consulted me March 7, 1894. He stated that he had always been of a sensitive disposition and inclined to be morbidly self-conscious. Of late years this had greatly developed and made his life a burden to him. He had the fixed idea that he was constantly making mistakes in business and that all those with whom he was brought in contact considered him a fool. During a business interview he was embarrassed and unable to speak and felt that everyone must notice this. He had the same feelings in reference to society and shunned it as much as possible. He also had morbid and entirely unfounded fears about his physical condition. Hypnotised ten times to July 11, 1894, when his morbid ideas had entirely disappeared. He has just reported himself, April, 1895, as well. If he has any anxieties about his business, he says, these are only natural ones and he has lost all his morbid fears and embarrassments. He is now fond of society and at his ease in it.

(9) Mr. I., aged 35, was sent to me by Dr. de Watteville on October 29, 1894. Illness commenced six months ago after the sudden death of his brother-in-law. He began to sleep badly and to dream of his own death. The insomnia continued and he was haunted by constant fears in reference to himself and his family. He dreaded that he would lose his employment and that he would find his wife and children dead when he returned from work, &c. He became afraid of open spaces and was unable to cross the road without assistance. One day, when sitting alone in his parlour, he believed he saw two men bring his coffin in. He never had a moment's freedom from his fears and was so miserable that he had strong suicidal impulsions. About this time he commenced to have frequent attacks of giddiness and felt that he would fall down if he did not catch hold of something. On one occasion only did he lose consciousness. He was hypnotised five times up to November 12, upon which date his morbid fears had almost entirely disappeared, but as he still had attacks of giddiness and sometimes felt depressed, I continued to hypnotise him

once or twice a week up to the present date (April 26, 1895). Present condition: He sleeps well, has had no giddy fit for two months, no agoraphobia or return of his other morbid fears, and deep hypnosis can now be easily and quickly induced.

(10) Mr. Watts. I was asked by Dr. Hack Tuke to try to hypnotise this patient, No. 10 in his list of cases. At this time he had been twelve months in Bethlem Hospital and was on the eve of being discharged, as his mental condition remained unchanged, and it was considered unadvisable to retain him longer. Dr. Percy Smith kindly consented to keep him another month for the purpose of this experiment, and I attempted to hypnotise him twenty-three times without success. I met him accidentally the other day; he told me his condition was unchanged and that he was going to live on a farm.

Dr. Hack Tuke also asked me to try to hypnotise No. 11 of his cases. When I saw this patient he was obviously insane and extremely suspicious, and nothing would induce him to submit to the treatment.

(11) [Liébeault.] Mrs. ——, May, 1892, aged 36. Has had good health until the present illness commenced eleven months ago. Her father committed suicide by drowning; her first husband was accidentally drowned twelve days after marriage, and she lost her second suddenly after she had been married three years. Since then she has avoided her friends, slept badly, neglected her work and her person, and has had the fixed idea that she would commit suicide by drowning as her father did. Hypnotised fifty-two times from May to July, 1892, when all ideas of suicide disappeared. In a recent letter Dr. Liébeault tells me that she remains well mentally and physically.

(12) [Schrenck-Notzing.] Mr. ——. Fixed idea regarding catching cold kept this patient for six months in his room. Hypnotised. Recovered.

(13) [Schrenck-Notzing.] Mr. ——, aged 24. Had periods of terror and uncontrollable sensations and ideas with the fixed idea that his *fiancée* did not love him. Hypnotised. Recovered.

(14) [Hecker.] Mrs. ——, suffered from diseased desire to wash herself continuously and unnecessarily; had morbid fears that the doors and windows were not properly fastened, and fancied that she dressed herself untidily. On one occasion, after having spent two hours vainly trying to dress, she sent for Hecker, who found her in a state of great anxiety, putting on and off her clothes until exhausted. Hypnotised for a month. Recovered.

(15) [Wetterstrand.] Mrs. ——, aged 42. For several years

had a morbid dread of thunder-storms; did not dare go into the country in summer, and in winter dreaded what the summer might bring. Constantly watched the appearance of the sky. Hypnotised twenty times. Recovered.

(16) [De Jong.] Mr. ——. Agoraphobia of many years' standing. Hypnotised ten times. Recovered. No relapse after a year and a half.

(17) [De Jong.] Mrs. ——. Fear of storms and of travelling by rail. Hypnotised. Recovered.

(18) [Van Eeden and Van Renterghem.] Mr. ——, aged 56. Psychical impotence for three years with the fixed idea, which he recognised to be false, that his wife wished to be unfaithful to him. Hypnotised eight times. Recovered. No relapse after twenty-two months.

This list might be largely extended. Many of the cases referred to at the commencement of the article were successfully treated by hypnotic suggestion, and many others have been reported by Bernheim, Delbœuf, Gorodichze, Russell Sturgis, Voisin, Burot, Mavroukakis, Bourdon, &c.

My chief difficulty in such cases has been the induction of the primary hypnosis. Various continental statistics apparently show that the majority of mankind can be hypnotised; Schrenck-Notzing's "First International Statistics of Hypnotic Susceptibility," published in 1893, give 8,705 cases by fifteen observers in different countries, with six per cent. of failures. Wetterstrand reports 105 uninfluenced out of 6,500 cases, and I formerly found patients drawn from my own practice equally susceptible.

For the induction of hypnosis the patient's attention is necessary, and in cases of imperative ideas, this is usually otherwise occupied as the result of the nature of the mental affection. Many also belong to the class the most difficult to influence, *i.e.*, the nervous and emotional. That such patients are the easiest to hypnotise is the conviction of all those who have no practical acquaintance with the subject, but this opinion, unfortunately, has no foundation in fact. Moll says: "The mentally unsound, particularly idiots, are much more difficult to hypnotise than the healthy. Intelligent people and those with strong wills are more easily hypnotisable than the stupid or weak-willed. Sex has no particular influence." According to Forel, "every mentally healthy man is naturally hypnotisable." Mr. Wingfield hypnotised 152 Cambridge undergraduates at the first attempt, while Esdaile found no difficulty in inducing hypnotic anæsthesia among the coolies and felons of Bengal,

and these, as he justly remarked, were by no means nervous persons. The fact that many of my patients had read sensational newspaper and magazine articles on the subject did not render my task easier.

Personally I have never seen the slightest untoward symptom result from the skilled use of hypnosis, nor any evidence that its dangers, under these circumstances, have any existence save in the brain of the novelist. Forel says: "Liébeault, Bernheim, Wetterstrand, van Eeden, de Jong, I myself, and the other followers of the Nancy School, declare categorically that we, supported by the material of many thousands of hypnotised persons, have never observed a single case of mental or bodily harm caused by hypnosis, but on the contrary have seen many cases of illness relieved or cured by it."

As regards the connection between "folie du doute" and "délire du toucher" it is interesting to notice that the latter condition occurred in No. 7 as soon as the patient's doubts took a material form, but was not associated with it when his obsessions were purely intellectual. Does not this show that the appearance of the "délire du toucher" simply depends upon the nature of the "folie du doute," and that the incessant washings, &c., are the patient's natural physical efforts to rid himself of his material fears, while the character of the purely intellectual obsessions renders such relief impossible. G—— sought and found its nearest equivalent in telling his religious troubles to an older friend, in whose opinions he tried to find consolation. From this point of view the second of the cases quoted by Ladame shows "folie du doute" unassociated with "délire du toucher." This separation is, I think, more artificial than real. In his first case, that of the young man who frequently washed his hands through fear of contamination, it is by no means clear that this was a case of "délire du toucher" without "folie du doute." Surely the abnormal and incessant washing must have been the result of doubts which he thus tried to remove. The same objections might be raised to van Eeden's cases where "folie du doute" is said to have resulted from "délire du toucher."

Dr. Hack Tuke regards imperative ideas, and the acts resulting from them, as essentially automatic and considers this their fundamental characteristic. Are these acts automatic? An automatic act is simply an habitual voluntary one performed inattentively or unconsciously; while the so-called automatic acts of the sufferer from imperative ideas are carried out in opposition to his volition and frequently associated with intense

and painful consciousness. Possibly with justice they might be called reflex, seeing that they are "fatal, unchosen, response to stimulation."

The fact that an imperative idea remains a stranger to the patient's ego distinguishes it, according to most authorities, from an insane delusion. This rule has its exceptions. One of my patients commenced ten years ago to be "inhibited" by various superstitions. At first this rarely occurred, but latterly, owing to the number of his unlucky days, &c., the performance of many acts is often interfered with. Many people, by no means insane, actually believe in, and are influenced by, identical superstitions. This patient, however, does not believe in them and keenly resents their interference with his actions. The non-assimilation of the imperative idea sometimes, then, constitutes the morbid element, and this apparently depends more upon the individual than upon the idea itself. The patient who made herself miserable about the Creation might, under other times and circumstances, have taken pleasure in discussing "the number of angels who could stand on the point of a needle, or whether, in passing from point to point, they had to traverse the intermediate space." The imperative idea to discover the site of ancient Troy only differed from those we are discussing in the fact that it was assimilated by its possessor, but this did not constitute insanity.

In nearly all my cases the condition appears to have had an emotional origin. The shock of the sudden death of a relative caused one patient to fear his wife would die; another dreaded travelling after being frightened by a drunken man in a railway carriage. Although in some instances the emotional element changed its character, and in all became greatly intensified, it was certainly often associated with the commencement of the original trouble.

Imperative ideas are usually regarded as being typical of degeneracy and especially of hereditary degeneracy. Some of my cases seem to confirm this, they were weak mentally and physically and had unsatisfactory hereditary antecedents. In several instances their imperative ideas had become insane delusions, many of them had suicidal impulses, one attempted suicide and another had hallucinations. On the other hand, the transition from the normal state to imperative ideas is almost insensible; the repetition of an insignificant saying being, according to Ribot, the slightest form, and pre-occupation, such as anxiety about an examination, a degree higher. Most children too have suffered at one time or another from imperative ideas.

This, as a popular writer has justly remarked, appears to arise from an exaggerated sense of the importance of what they say and do, and also from an exaggerated fear regarding the notice taken of them by others. He says: "How miserable we sometimes make ourselves over some silly remark we have made. Some of us even keep a little store of foolish things we have said or done at various times—and take them out occasionally and blush over them. As a child I blushed for years at the thought of having piped out a response in church in the wrong place, before the clergyman's turn was over. I felt as if the whole congregation turned and gazed at me with scornful ridicule. As I walked away everyone who glanced at me I felt sure was thinking, "There goes the child who made that extraordinary squeak in church."

Everyone cannot have fixed ideas, as for example idiots, who possess little spontaneous and no voluntary attention, while, as Ribot says: "In every sound human being there is always a dominant idea which regulates his conduct; such as pleasure, money, ambition, or the soul's salvation." Some of my patients were physically far above the average, and many of them possessed mental endowments of high quality, and their morbid ideas did not prevent them doing valuable work. Most of them, it is true, were of an emotional, nervous type, but is the sensitive, mobile brain necessarily degenerate? May not the accidents to which it is liable be the result of its higher and more complex development? The thoroughbred is more emotional and nervous than the cart horse, but is this necessarily an evidence of its hereditary degeneracy? The term degenerate is applied so freely and widely by some modern authors that one cannot help concluding that they rank as such all who do not conform to some primitive, savage type possessing an imperfectly developed nervous system.

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J. MILNE BRAMWELL.

ON SYPHILIS OF THE NERVOUS SYSTEM.

[With a Critical Digest.]

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(*Continued from p.* 147w.)

APPENDIX TO PART III.

THE third part of this communication, published in the last number of BRAIN,[1] dealt with syphilitic disease of the spinal cord. It happened to be published a few days before Mr. Jonathan Hutchinson[2] brought forward the discussion of cases of nervous disease occurring early in syphilis, with especial attention to cases of syphilitic paraplegia during the secondary stage, or during what was assumed to be so. Incidentally, the *early* occurrence of syphilitic spinal cord disease had been the subject of considerable notice in my communication; as, *e.g.*, where syphilitic disease of large spinal veins and arteries, with the accompanying meningitis—which so often is of gummatous form—were taken "to explain such occurrences as local softening, hæmorrhage, sudden increase of pre-existent paralysis or paresis, or the onset of sudden paraplegia—perhaps at an early stage of the disease—perhaps even appearing almost like a precursory phenomenon" (p. 144). Or where it is stated that many cases of syphilitic paraplegia, sudden or rapid in onset, attributed to myelitis, are probably due, in reality, to the softening of portions of spinal cord following upon occlusion of syphilitic blood vessels. Or where sudden flaccid paraplegia was found to be due to hæmorrhage into the spinal grey from syphilitic blood vessels with lepto-meningitis. Or where Gilbert and Lion's investigations of the pathological changes in early syphilis of the spinal cord are stated in sum-

[1] BRAIN, Spring Number, 1895, p. 141.

[2] Roy. Med. Chir. Soc., London, February 26, 1895.

mary. Or where the paraplegia is described as being, "less often severe acute palsy, perhaps from arterial thrombosis or from acute central myelitis, which usually is promptly fatal," and the softening or death of portions of the cord is described as being sometimes rapid or sudden.

Mr. Hutchinson brought fifteen cases of acute paraplegia occurring between half-a-year and two years after the primary syphilis. But other similar cases which he observed occurred later after infection. In general type, apparently, they only differed somewhat from Erb's; and did so in the rapidity or suddenness of onset, or of development, of the paraplegia, and the frequently severe degree of anæsthesia occurring at least temporarily. Like Erb in *his* cases he assumes them to be due to transverse dorsal myelitis, but had had no deaths. The cases with necropsies, cited from Gull, Fagge, Arthur Fox, Cropley and others, showed chiefly an acute myelitis.

In the discussion, Dr. George Ogilvie set forth a statistical array of examples of *early* nervous affections in syphilis, drawn from the writings of continental authorities.[1] Of these the most striking and most relevant to the point under discussion, is the experience of Goldflam, who "amongst eighteen cases of syphilis of the spinal cord found thirteen during the first two years." And Dr. Ogilvie rightly added that "cases of exactly the same nature, indistinguishable from the early ones in any way, occur late in syphilis."

Differing from the general type made by Erb, as well as from the more often acute (compared with Erb's) but non-fatal cases by Hutchinson, there is a severe and often fatal form of paraplegia. Usually an early phenomenon, probably it is not always necessarily or essentially so, as it has been stated to be by Lamy.[2] Most often, according to him, it occurs within, or shortly following the first year after the appearance of hard chancre. Most often sudden in onset, the paraplegia is generally heralded by a short premonitory stage; such as pain in the spine, weariness, or paresis, or bladder symptoms. The paralysis may come on in a night, in a few hours, or suddenly. When the disease has formed, the intensity of the symptoms is unusually great; absolute paralysis of motion, and even of sensation, may occur in the

[1] Braus, 14 per cent. of brain syphilis within first year after infection. Mauriac, 43 per cent. of cerebro-spinal syphilis within two years. Rumpf, 23 per cent. of brain and cord meninges syphilis within one year. Goldflam, 72 per cent. of spinal syphilis within two years.

[2] "De la Méningo-Myélite Syphilitique, p. 101."

parts affected, although not in all cases, for the anæsthesia may be incomplete or may be unilateral. The tendon-reflexes are diminished or abolished and then the limbs are flaccid. Electro-muscular excitability may be lessened or lost, paralysis of bladder and rectum may be complete. Bed-sores tend to form. The usual lesion is an acute central myelitis. Or there may be luetic spinal lepto-meningitis, chiefly affecting the veins, and partly implicating the cord itself, in which latter, also, are secondary changes due to interference with the circulation, and consisting of colloid exudation in the grey, and areas of nerve-fibre degeneration in the white (Hayem, Julliard, Siemerling, Déjerine, Erb, Lancereaux and others).

PART IV.

Syphilis and Tabes: their Relationships.

A.

The doctrine of the syphilitic origin of tabes dorsalis is mainly due to Prof Fournier,[1] of Paris, and to Prof. Erb,[2] of Heidelberg. But thirty years ago, in his work on tumours, Virchow asserted that, indubitably, many cases of tabes dorsalis—of grey atrophy—are syphilitic. Also, previously to Fournier and Erb, had Wunderlich mentioned, besides permanent paraplegias, tabetiform symptoms as due to spinal syphilis; and Berger[3] appears to have observed much the same thing.

Lancereaux[4] admitted the possibility of at least something analogous to tabes as a result of syphilis.

Erb,[5] in 1883, and in part previously (*loc. cit.*), declared syphilis to be one of the most important, or *the* most important of the conditions for the origination of tabes, and that a person not previously affected with syphilis has scarcely any chance of becoming tabetic. And, in 1892,[6] he gave 89 per cent. of tabetics as having had previous syphilitic infection, as compared with twenty-

[1] "De l'ataxie locomotrice d'origine syphilitique," Paris, 1876.
[2] *Deutsches Archiv für Klin. Med.*, July, 1879.
[3] Cited by Prof. A. Eulenberg.
[4] "Treatise on Syphilis," *New Sydenham Soc. Trans.*, 1869, vol. ii., p. 86.
[5] *Berliner Klinische Wochenschrift*, 1883, No. 32.
[6] *Berliner Klinische Wochenschrift*, 1892, Nos. 29 and 30.

two and a-half per cent. of other hospital patients who were not tabetic. Leyden,[1] in 1883 and 1892, took the opposite view.

But while Erb did not consider it proved that tabes is a specific syphilitic disease, or late manifestation of syphilis, Moebius[2] (1884) held empoisonment of the organism as necessary to tabes, admitting as the only efficacious poison—syphilis; and, doubtfully, ergot.

So long ago as 1876, writing on syphilis and insanity, in the part of the article devoted to gross syphilitic nervous disease, I published, amongst other cases, one, the spinal element in which was spoken of as simulating tabes. (There were typical ataxy, Romberg's symptom, &c., and intercurrent strabismus and diplopia, also dimness of vision.) And it was so spoken of because, at that date, syphilis was not, or was scarcely recognised as in any direct ætiological relationship with tabes, *i.e.*, as productive of "systematic" spinal cord disease, and the mental recovery (at least for the time being) and the vast improvement of the tabetic symptoms, under anti-syphilitic treatment, led me, at the time, to conclude that the symptoms were scarcely due to true tabes but to ordinary syphilitic inflammation or neoplastic diffuse growth. *Now*, some would say it was a case of tabes, and probably it was the first case published in this country as being syphilitic.

The question of recovery, in this and similar cases, will recur after mention of two other cases of well-marked, typical, tabetic symptoms, in persons believed to have had previous syphilitic infection, on whom I made necropsies some years ago. Both were general paralytics. In one there was chronic spinal meningitis and thickening, particularly over the posterior surface of the cord. The posterior columns presented the typical grey degeneration and sclerosis of tabes, chiefly in the lower dorsal and lumbar segments of the cord, in parts of which the whole of the posterior median and posterior external columns was affected, but, above, the lesion gradually becoming limited to the posterior median columns, and invading the bulb. Many of the lower posterior spinal nerve roots were similarly lesed. Some optic nerve atrophy.

In the other, also, was chronic spinal meningitis, all the changes about to be mentioned being confined, or nearly so, to the posterior surface of the cord; namely, thickening of spinal dura

[1] "Real-Encyclopädie der Gesammten Heilkunde," 1883. *Berliner Klinische Wochenschrift*, 1892, Nos. 17 and 18.

[2] Cited by Eulenberg.

[3] *British and Foreign Med. Chir. Review*, July 1876, p. 194-5.

and arachnoid; numerous white cartilaginoid plates in the layer of arachnoid next the cord; numerous adhesions of the meninges between themselves and to the cord, by distinct pseudo-membranous bands and filaments. The arachnoid thickened, opaque, semi-gelatinous in appearance. The posterior columns of the cord were lessened in consistence, altered in appearance, and the seat of myelitis taken to be of subacute type (numerous round cells, infiltrating profusely. Some scattered granule-masses).

Interesting in relation to the mobile recurrent right external strabismus (and to [?] the permanent slight right exophthalmos) was the subacute inflammation found affecting part of the length of the right motor oculi nerve near its root.

B.

Reverting to the recovery of apparent cases of tabes as bearing upon the question of their syphilitic origin, it has been held (as, *e.g.*, by Gowers,[1]), that as the symptoms of syphilitic disease of the nervous system depend, not on the specific process, but on the alterations it produces in nerve tissue, and as treatment exerts a direct effect on the specific process only, it follows that the persistence of symptoms, in spite of treatment, indicates persistence only of the damage done to the nerve elements, and not a continuance of the lesion producing the damage.

But dissipation of the specific process must in many, especially in early, cases permit the damage of nerve elements, still incomplete, to be repaired, or partly so; and it has been reported by Dinkler[2] that of Erb's cases of tabes 58 per cent. showed some improvement under anti-syphilitic treatment. And I have found tabetic symptoms somewhat improved, and the associated mental disorder (for which they came under care), alleviated under iodides. Sachs, also,[3] has found vigorous treatment with Hg. and iodides benefit the ocular, ataxic, tendon reflex, and painful symptoms of tabes. And going back to my early student days, although we saw tabetic patients treated with nux vomica, cannabis indica, anodynes, arg. nitr., and pot. iod., with or without actual cautery or blisters over the spine, yet a renowned clinical teacher, Da Costa, taught us that the only drugs at that time apparently of any ameliorative effect on the disease—as distin-

[1] "Syphilis of the Nervous System," 1892.
[2] *Berliner Klinische Wochenschrift*, 1893.
[3] *New York Medical Journal*, Jan., 1894.

guished from mere relief of pain, &c.—were iodide of potassium and nitrate of silver, so that the iodide is a very old friend in the treatment of tabes.

Some cases have been reported of improvement, of great improvement, or even cures of tabes[1] by anti-syphilitic treatment. For obvious reasons this is more likely to occur in private than in hospital practice. The patient is the more entitled to the chance of obtaining the possible benefits of specific treatment (Gerhardt) the nearer the onset of tabes is to the date of primary infection, the stronger and better nourished he is, and the more manifest are signs of syphilis. Moreover, the expectations of improvement under specific treatment are more favourable in the so-called anomalous or atypical examples of tabes, *e.g.*, cases with symptoms predominating on one side, presenting a less tardy course than the typical cases, and advancing by strokes. And Sachs suggests as a point sometimes helping to indicate cerebro-spinal syphilis simulating tabes (and, therefore, more curable than tabes proper)—an unusual irregularity of development of the symptoms; *e.g.*, the light reflex being lost in one eye very much earlier than in the other, or while the one is lost the other remains normal for years, or one or both pupils respond neither to light nor in accommodation, or any active syphilitic process at the brain-base is apt to be associated with or to long precede genuine tabes.

Are the alleged cures of tabes really examples of recovery from so-called pseudo-tabes? Syphilitic disease of spinal meninges with tabetiform symptoms may be accepted as one form of pseudo-tabes. And in "atypical" cases of tabes, instead of merely the ordinary typical tabetic sclerosis or grey degeneration of posterior columns and nerve roots, there may be a fresher and specific syphilitic morbid process.

Dr. Adamkiewicz has had several cases, a very careful study of which has led him to the belief that there is a special form of tabes having certain characteristic symptoms, and a specially defined clinical course, which is positively curable by *early* anti-syphilitic treatment.

"The symptoms of this curable form of locomotor ataxia are: Ataxic paresis with loss of the tendon reflexes, paresis in the distribution of certain cerebral nerves, tabetic paræsthesia without objective disturbances of sensibility, and pain especially on pressure in the lumbar region. The clinical course of the disease

[1] Cure in one case lasting 18 years (Reumont).

is characterised by a rapid advance of the paresis of the lower extremities into complete paralysis, by ascent of this paralysis up to the lower part of the cervical spine, and, when the anti-syphilitic treatment is early begun and energetically carried out, by a final complete restoration to health. The duration of the disease, when so treated, is three or four months. The most important clinical symptoms of the disease—ataxia and motor paresis—point to an involvement of the pyramidal tract as well as of the posterior columns, and the fact of a cure by means of iodides and mercury, to functional disturbance resulting from a not irreparable process. There can be question here of syphilitic products only, which appear under the form of obliterating end-arteritis and syphilitic pachy-meningitis.

"When, through lack of proper specific treatment, there is no resolution of the syphilitic products in the spinal cord, the diseased vessels become obliterated, and the parts of the cord supplied by them become sclerosed. In this case, similarly to what he has shown to occur in tuberculous pachymeningitis, wedge-shaped lesions are formed in the substance of the cord in which the nerves are destroyed. He has called this lesion, which results in incurable spinal paralysis, infarct of the cord."

The general belief that syphilitic vascular lesions of the brain only, and not of the spinal cord, are curable, is disproved, he said, by his observation.[1]

It is probable that most of us have erred in giving insufficient doses of iodides in syphilitic nervous diseases when heroic doses are needed. In cases of general paralysis and of tabes, apparently springing from syphilis, large quantities of K.I., or of Na.I., are given by some.

For example, Max Weiss[2] describes a case of advanced tabes, in which the connection between that disease and syphilis was very clear. "This case is especially noteworthy from the fact that a regular and steady specific treatment markedly and rapidly diminished pronounced objective and subjective tabetic symptoms, a few of these even disappearing entirely. The treatment consisted solely of rather large daily doses of iodide of sodium, increasing from 5 to 8 grammes (1¼ to 2 drachms) for several months. The patient was an engineer, 35 years old, who had never suffered from illness during childhood; in 1883 he

[1] Int. Med. Cong., Rome, *Medical Record*, May 5, 1894.

[2] *Centralblatt fur die gesammte Therapie*, February, 1894. Abstract from *Univ. Med. Journ.*

acquired an indurated specific ulcer, with secondary symptoms. He was given twenty injections of corrosive sublimate and small doses of iodide of potassium; in July, 1884, a lingual ulcer developed, which underwent complete resolution after twenty-four injections of corrosive sublimate. Since that time there had been no specific eruption either on the body or the visible mucous membrane. In 1886 several attacks of nausea and vomiting occurred, each lasting about fourteen days, accompanied by severe pain in the back. In the autumn of 1887 renewed attacks of vomiting occurred early in the morning (gastric crises). From 1889 there were almost daily attacks of vomiting. Nutrition was much impaired and the body-weight decreased. He suffered from lancinating pains over the entire surface of the skin, more particularly in the arms and legs, most frequent after a change of weather." In 1890, static and dynamic inco-ordination was first noticed, with paræsthesia of the toes, soles of the feet, and the two small fingers of each hand, diminished tactile sensibility in the epigastric region, and fatigue, after the least attempt at walking. "Constipation, cramp-like pains in the abdomen (intestinal crises), some retention of urine, and severe boring pains in the urethral canal were added to the other symptoms. In the spring of 1893 the sight was poor at a distance of from twenty to thirty steps, but there was no trouble in reading and writing. In August, 1893, the patient was submitted to a systematic iodide treatment. For the first two weeks he took daily 5 grammes (1¼ drachms) of diluted iodide of sodium, no symptoms of iodism being observed. The daily dose was increased 2 grammes (31 grains), and for some time 3 grammes (46 grains). Within a month the daily gastric crises ceased suddenly, and have never since reappeared; in September disturbances of co-ordination diminished perceptibly, and in October, when the patient was taking 8 grammes (2 drachms) of iodide of sodium daily, and had already consumed 500 grammes (1 pound) in all, without any untoward symptoms, no evidences of ataxia were present.[1] The cloudiness of vision had also disappeared, the lancinating pains occurred but seldom, and were much less severe than formerly. The patient, even after walking several hours, did not feel any fatigue. His appetite has greatly increased, and his weight has increased 6 kilos (12 pounds). Treatment is still being continued in daily doses of from 6 to 8

[1] Purity of the iodide of sodium employed is an essential condition of such treatment.

grammes (1½ to 2 drachms), with short intermissions. The paræsthetic symptoms have almost entirely disappeared." The urethral crises and the weakness of the detrusors persisted longest, and galvanization through the lumbar cord, perineum and bladder, was resorted to, with internal administration of ergot and strychnine, the result being that for three weeks before the time of report the patient no longer complained of urinary disturbance or of pain in the urethra.

Weiss refers to a similar case treated by Werner Stark. The patient was a woman, aged 43, who had become infected thirteen years previously and recovered without relapse. Eight years later the first indications of tabes appeared, the symptoms increasing in intensity until the patient was unable to walk. "No disturbances of the digestive or urinary organs occurred. When the patient was first seen by Stark, in 1890, she was pale and thin; there was complete ataxia of the legs, analgesia and partial anæsthesia of the skin, as well as weakness and atrophy of the muscles of the legs; the patellar reflex was absent. Psychic and ocular disturbances were not present. The patient had been discharged from hospital as incurable. Stark first gave 50 grammes (1½ ounces) in 400 grammes (13 ounces) of water, a tablespoonful being taken three times daily. After some time the pains became less severe." The solution was then strengthened to 60 in 400 grammes (1¾ to 13 ounces); after three months to 75 in 400 grammes (2¼ to 13 ounces); and again after three months to 100 in 400 grammes (3¼ to 13 ounces), four tablespoonfuls daily, this strong solution being taken for four months. "After the first increase of the dose improvement soon occurred, so that the patient was able to do a little light work; after the second increase the pains disappeared and the ataxia and anæsthesia decreased. After the last increase, when the patient was taking 12 grammes (3¼ drachms)[1] of the iodide of potassium daily, there was perceptible improvement; the anæsthesia and ataxia disappeared, and the muscular atrophy diminished; the patient could walk quite well with the aid of a cane or support. During the entire time she did not suffer from any disturbances of the digestive or other organs. Sometimes the treatment was continued steadily for weeks, and again it was interrupted at intervals; during these, however, Stark observed that improvement was not progressive, and that there was occasionally a tendency to relapse. At the time of writing, the patient had, for a year only, occasionally been taking the iodide of potassium, feeling stronger after each treatment."

[1] Fifteen grammes (4 drachms)?

C.

From the third Part of this communication, namely, that on syphilitic spinal cord disease generally, we carry forward a reminder of the cases linking tabes with spinal cord disease clearly due to the specific lesions of syphilis. Such, especially, are the cases of syphilitic lepto-meningitis invading the cord, particularly its posterior columns and horns, sometimes producing a more or less tabetic, or quasi-tabetic, posterior sclerosis, and diagnosed during life as examples of tabes, or of anomalous, atypical, or pseudo, tabes. Of such kind are cases therein cited from Ewald, Eisenlohr, Kuh, Dinkler, Brasch, Schulz, Siemerling, and one or two of the present writer's cases.

Moreover, syphilitic spinal lesions have been found associated with the recognised lesions of typical or classical tabes. In this communication (see Parts I. and III.) have already been described in abstract two such cases, one from Raymond, and one from Hoffmann, and the second of the present writer's three cases, mentioned in this Part IV., was probably of that nature.

Here another may be added from Sachs.[1] "Clinically it was a typical example of tabes dorsalis."

Posteriorly, the spinal pia was covered by a thick gelatinous mass, from lower cervical to upper lumbar segments. The pia was thickened both in front and behind, and of leathery consistence. Microscopically, was diffuse sclerosis of posterior columns, extending from the lower cervical region to the lumbar enlargement, but best marked in the cervical and dorsal segments, and it included Lissauer's tract. In the lumbar segments, only the posterior portion of the posterior columns was affected much, the rest but little. "A few cells of Clarke's column could be made out only in the lower dorsal segments. The sclerotic tissue differed in no respect from the classical order of spinal sclerosis." Nerve fibres of cord elsewhere, and anterior grey matter, normal. In lumbar and dorsal segments was a widespread meningeal infiltration; also, there, the pia was firmly agglutinated to the cord, particularly in the posterior and lateral column regions; there, also, the posterior nerve roots were degenerate, and the small blood vessels showed changes characteristic of syphilis. These changes were most in the lumbar region, where the sclerosis, on the contrary, was least. The pia was "characterised by an invasion and proliferation of round cells, and by very typical changes in its blood vessels, large and small."

[1] *New York Medical Journal*, January 4, 1894.

Consequently, besides posterior spinal sclerosis was a syphilitic lepto-meningitis "invading the cord at different levels, and occasionally invading a distinctly sclerotic area, also a very typical specific arteritis. Whether it was an endarteritis or a mesial arteritis cannot be determined easily," but in the majority of the vessels the innermost coat had undergone the greater changes.

The condition was taken to be a syphilitic process, superimposed upon the sclerosis, the sclerosis coming first, the syphilitic infiltration later on. So far as it invaded areas already occupied by sclerosis it would not occasion new symptoms. It is possible, or even probable, that the blood vessel change preceded the sclerosis.

A case of typical tabes dorsalis with central gliosis, in a syphilitic subject is reported by Nonne.[1] Besides the lesions of both conditions, were a recent focus of softening, explaining the paralysis of upper limbs; old ulnar neuritis; chronic cerebral lepto-meningitis; syphilitic endarteritis and mesarteritis of basilar.

In reference to cases of these kinds, besides what has already been stated, we shall notice presently that not only may myelitis or gumma be followed by tabes, but also that myelitis may come on in the course of tabes.

Clinically, also, we, in fact, get at least a close approximation to, perhaps an identity with, several clearly defined clinical entities according to the part or parts of the cord predominantly affected by syphilitic lesions. If these parts are the posterior columns (nerve roots and horns), the clinical image of tabes presents itself. If the postero-lateral columns (including crossed pyramidal tracts) are chiefly affected, and the posterior horns also somewhat invaded, the posterior columns less affected, the posterior root zones and nerve roots suffering little; then we get the symptom-group of Erb's so-called "syphilitic spinal paralysis." If the posterior, pyramidal-tract, part of the lateral columns chiefly suffered, there would be a symptomatic appearance as of primary spastic spinal paralysis, but in such cases other degenerations would perhaps be present, as may be in ordinary primary spastic paralysis, namely in Goll's and Burdach's columns, and direct cerebellar tracts.[2] If, as occurs rarely, the anterior horns suffer most, there are, with other symptoms,

[1] *Archiv für Psychiat.*, Band, xxiv. H. 2.
[2] Ad. Schule, *loc. cit.*

the phenomena of spinal progressive muscular atrophy, or, more acutely, anterior poliomyelitis; perhaps or possibly still more acutely, that form of it with, clinically, the acute ascending spinal paralysis (of Landry); if, with the degeneration of nerve cells of anterior horns, there is also predominant change in the lateral pyramidal tracts, the clinical aspect will be that of amyotrophic lateral sclerosis. Syphilitic cervical spinal cord change may simulate syringomyelia or Morvan's disease; and the syphilitic incidence chiefly on many of the posterior spinal nerve roots occasions a special symptom-group, as in cases by Kahler and Buttersack.

Likewise, the image of ataxic paraplegia may be the manifestation of syphilitic disease affecting both the posterior and the lateral columns, although such cases, finally, are apt to pass into a more spastic phase, and present the symptom-group of the spastic condition, already fully discussed in the Third Part of this communication.

But usually the lesions are multiple, partly diffuse, and partly concentrated in place, and the symptomatology correspondent thereto is mixed, combined, multiple; the disease being more or less widespread, yet selective of loci—selective, yet having a variety of affinities or tastes, so to speak.

In some of these cases of doubt whether there is genuine tabes; or, on the other hand, spinal syphilis, that is to say, in the sense of syphilitic morbid processes; the symptoms point to tabes, but there occurs a return of the knee-jerks which had been lost, or a restoration of equality between them on the two sides which had failed; and in some instances a cessation of external ocular paralyses, which had previously accompanied the failure in patella tendon reflexes.

The fact is also to be borne in mind that certain cases of tabes (contrary to what has been held by some) present not only the typical lesions, but also degeneration of the comma-shaped bundle more distinctly than of the surrounding parts. Or cases may present degeneration of the antero-lateral ascending tract; or of the direct cerebellar. In fact, the complications attending, sometimes, the lesions of genuine tabes are numerous.

D.

We may now turn to the studies of some observers on the proportion, per cent., of tabetic cases presenting a previous history, or indications of syphilis.

Dr. Gowers,[1] in private consulting practice, found 58 per cent. of male cases give a history of hard chancre or of secondary syphilis. Two-thirds, he thinks, would be nearer the truth, could all facts be ascertained. In women he found antecedent syphilis to be traced rather less frequently than in men. "In the cases of tabes that succeed syphilis, the lesion is not syphilitic in histological character. In these, as in the cases in which syphilis can be excluded, the disease is a degenerative one. It is, moreover, rarely influenced by anti-syphilitic treatment. Hence it must be regarded as a degenerative sequel of syphilis rather than as a true syphilitic disease. . . . In a few cases, perhaps 10 per cent. of the whole, syphilis can be excluded with confidence. The causes operative in these can be traced also in some of the patients who present a history of syphilis, and in such cases the causation of the malady is probably complex."

Such other causes are spinal injury, exposure to wet and cold, excessive fatigue and over-exertion, acute diseases, *e.g.*, rheumatism and enteric fever, alcoholic excesses.

In the second case of tabes ever seen by the writer, excessively hard work and over-exertion, six years previously to the time the patient was seen, were apparently the (or the chief) ætiological factors.

Fournier has stated that over 90 per cent. of persons suffering from tabes have incurred syphilis. Yet in only 76·7 per cent. of these (112 in 146) was antecedent syphilis alleged to be indubitable. On the other hand, Borgherini[2] has recently collected statistics in Italy, according to which scarcely 30 per cent. of those with tabes were syphilitic. In A. Fraenkel's experience, the percentage was 50·7 per cent.; in Senator's and Mendel's, 70 to 75 per cent. G. Lewin denied these high percentages. Virchow considers the relations of tabes and syphilis not yet decided, and notes the rarity of visceral syphilis in tabes.

Of 102 cases of tabes, Gerhardt[3] concluded that 51, or exactly half, were persons previously syphilitic. The *average* interval between the time when syphilis was incurred and the onset of tabes in these cases, was found to be ten years; from collected statistics, another places it at eight and a-half years.

Compare with this the *average* interval of only four years, in examples of spastic spinal paralysis, taken to be syphilitic.

Yet the same observer speaks of seeing syphilitic spinal cord

[1] "Diseases of the Nervous System," vol. i., 1886, p. 288.
[2] *Medical Record*, May 5, 1894. [3] *Loc. cit.*

disease supervene at from three months to twenty years after primary infection, and take similar forms under such very dissimilar time-circumstances.

On the other hand, in 17 cases of insular sclerosis, no mention of syphilis was found.

Prof. A. Eulenberg[1] discusses "syphilitic tabes" in the pathologico-anatomical sense, *i.e.*, the occasional appearance of syphilis of the spinal cord under the clinical form of tabes dorsalis. He published brief particulars of 125 cases of tabes diagnosed with certainty, all other examples, he says, being excluded. Of these, 19 were women; for reasons given, he excepts them from consideration, as to the ætiological factors; but it is to be noted that in only one of them was syphilis certain; a few others were sterile, and one had a husband probably tabetic, points which raise a suspicion of syphilis. But the majority of the women were certainly not syphilitic.

Of the 106 male cases, 39 had some venereal infection; but of these, 11 had had only simple or "soft" venereal sores or chancres, without subsequent appearance of general syphilis. These, I think, we must exclude. The remaining 28 had had typical hard chancre, and general constitutional syphilis. These we may accept as syphilitic, and these only. Thus, for male tabetics there would be 26½ per cent. (nearly), previously affected with syphilis. (The percentage would be 36¼, if we include, as Eulenberg does, the eleven cases with simple, soft venereal sores, and no subsequent external, or constitutional or general manifestation of syphilis.)

Neuropathic heredity was observed in 12 per cent.

Atmospheric *noxæ* (chills, wettings, &c.), in more than one-third.

Severe bodily exertions in one-fifth.

Severe depressing emotions, previously, in 13 per cent.

Debilitating conditions, leading to general weakness and exhaustion, in 10 to 11 per cent. (*e.g.*, pulmonary, intestinal, ovario-uterine, or malarial diseases; sexual and alcoholic excesses).

Traumatic damages in about 5 per cent.

Deformity of vertebral column in a few cases.

Previous acute disease in 5 cases (typhoid fever 4, dysentery 1).

Very often two or more of the above alleged factors were supposed to have acted conjointly.

Taking persons affected with all forms of nervous disease,

[1] *Archiv. für pathol. Anat.*, Bd. xcix., Hft. 1, p. 18.

collectively, Minor[1] found a syphilitic history about five times as frequent in Russians as in Russian Jews. In the latter, syphilis is very rare, owing to alleged reasons, which are duly set forth. And he found tabes about five times per cent. more frequent in Russians than in Russian Jews, and general paralysis about seven times more frequent. And yet to nervous diseases generally, the Russians are *less* obnoxious than are Russian Jews.

In eight tabetic women he found seven certainly syphilitic, and the remaining case probably so.

The combined statistics in Russia (his, Korsakoff's and Kojewnikoff's), if correct, show that, among those affected with various nervous diseases, there were:—

In 4,700 Russians, 137 cases of tabes, or 2·9 per cent. (29 per 1,000).

In 696 Russian Jews, 6 cases of tabes, or ·86 per cent. ($8\frac{6}{7}$ per 1,000).

These combined statistics thus show the racial proportion, per cent., to be only about (not 5, but) $3\frac{1}{3}$ times greater in the former than in the latter race.

Of the Russian tabetics, syphilis was believed to have existed in 60 per cent., for certain; and probably in some more. Of the eight Russian Jewish male tabetic cases he had known of (partly in above statistics), seven are stated to have been syphilitic.

The time between primary infection with syphilis and the onset of tabes, was found by A. Eulenberg to vary from either one or two years to twenty-nine years. Isaac cites an interval of from one to twenty-five years.

The last named[2] gives importance to the old objection that, whereas tabes is a system-disease of the cord, syphilitic disease, on the contrary, is characterised by interstitial or tumour-forming processes, so different from the primary parenchymatous morbid processes affecting the nerve elements in tabes, and holds that the statistics have too many sources of error to attain certainty on the ætiological connection between syphilis and tabes; that there is not an ætiological relation between them; but syphilis, like other influences leading to tabes, may act by enfeebling a disposed nervous system; and that anti-luetic treatment is contra-indicated in tabes.

Too often, we think, the statement of a pre-existent syphilis, or even a simple venereal sore, real or supposed, has been taken

[1] *Archives de Neurologie*, t. xvii.

[2] *Berliner Klinische Wochenschrift*, April 16, 1894.

as sufficient proof of the existence of syphilitic infection, and, impliedly, of the syphilitic origin of a subsequently existing tabes. The same objections hold here as do also regarding the similar statistics in relation to general paralysis (see Part I.).

Some of the errors into which observers and compilers of statistics have fallen, in this matter, are referred to by Hr. G. Lewin.[1] One is that genital scars have been taken as indication or proof of antecedent syphilis; whereas a soft venereal sore usually leaves a cicatrix, but a true hard one (sclerosis) only does so very rarely.

Moreover, and this is another source of error, a soft sore may become indurated, and such non-syphilitic hard (hardened?) chancres he designates, in their several kinds, as (1) follicular, (2) cicatrised, (3) artificial, and (4) phlegmonous. It is unnecessary to point out how misleading, therefore, may be a history of a hard sore.

Another error concerns the diagnostic difficulties as to the rashes, the ophthalmias, &c., of syphilis.

Following Virchow, he has found atrophy or aphasia of glands of the tongue, a valuable indication of syphilis in several cases where other specific lesion was scarcely to be found.

Blaschko emphasizes the importance of examining the patient's children when the personal examination fails to reveal any clear indication of tertiary syphilis, such as gummatous orchitis or smooth atrophy of tongue-root. In the children, "Hutchinson's teeth," and similar dental anomalies, are important for diagnosis of the parental malady.

E.

Is the morbid process in tabes proper due to syphilitic infection, including here, not only the spinal, but also the cerebral elements of tabes?

The posterior horns and columns, including Lissauer's tract and the root zones in postero-external columns, in which tabic changes first appear are quasi-accessories to the spinal cord, and represent, quantitatively and qualitatively, the entrance of posterior root fibres at the various levels of the cord. So that, in one sense, tabes appears to be a degeneration of posterior roots as they enter the grey and white of the cord.

As to the question whether this degeneration is *primary* or

[1] *Berliner Klinische Wochenschrift*, 1894, p. 657.

not, Sachs reminds one that degeneration of nerves occurs if they are cut off from the influence of their trophic nerve cells. For the sensory posterior nerve roots and fibres, these cells are in the spinal ganglia on posterior roots, and in the terminal nervous end organs, chiefly in skin and musculature. Sufficient disease in those ganglia or in these bodies, or lesion of the nerves in their course, may give rise to the degeneration; ascending degeneration of sensory fibres in the cord would therefore follow (*inter alia*) a lesion between the spinal ganglia and the posterior horns of the cord. This lesion may be set up by syphilitic spinal meningeal disease: explaining a relation between the morbid anatomy of syphilis and of tabes. Thus further questions arise: (*a*) whether the syphilitic poison acts through the medium of the blood vessels, (*b*) whether the syphilitic poison brings about changes in the blood, and, if so, (*c*) whether these hæmatic changes are the cause of the changes in the walls of the blood vessels.

Ergot, a potent agent on the vascular system, also occasions a so-called form of tabes.

F.

We must now briefly refer, more pointedly, to the intoxications of the organism—its toxic or toxæmic states—whether self-produced, or coming from without—whether chemical, or the results of perverted metabolism, or of microbic origin. These states are apt to manifest themselves by organic changes in the nervous system, and preferably by alterations either in the peripheral nerves, or in the posterior spinal columns, or in both. Thus, posterior columnar spinal changes are found in many infectious microbic diseases; in some maladies consisting of alterations in the constitution and life of the blood, however these may be produced; or consisting in perversions of metabolism, and of excretion; or are found in systemic intoxications which are to be directly connected with prejudicial drinks and foods.

Thus, like the peripheral nerves, the posterior columns appear to be particularly vulnerable in toxæmic, or exhausted cachectic states.

As to the relations between syphilis and tabes, and the question of toxæmia, the former are brought out more clearly in cases of tabes in the upper classes of society, in gentlewomen, and in children with hereditary syphilis.

If we admit a connection between syphilis and tabes, of what kind is it? That, at present, we cannot accept tabes as simply

or absolutely a tertiary syphilitic disease is the view of Professor Strümpell;[1] and that to form a more exact conception of the kind of connection will assist one to meet objections raised against the very existence of a connection. The most important of these objections are two, namely: (1) first, the often alleged total or comparative failure of anti-luetic treatment to control or to cure tabes; and (2) second, that the pathological anatomy of tabes is not akin to genuine syphilitic changes, but fundamentally represents a simple degeneration of some nerve fibre tracts. It is unsatisfactory to meet these objections by over-estimating the effect of anti-luetic treatment in tabes, and by deducing the morbid anatomy of tabes from vascular disease. A more satisfactory basis on which to take stand, is the recognition that tabes after syphilis would form no isolated phenomenon in pathology, but would fall into the large group of nervous consecutive diseases or lesions following infections of the most different kinds. For, after diphtheria, typhus, dysentery, and many similar infections, degeneration may affect definite portions of the nervous system—degenerations which are secondary simple degenerative processes, and not specific primary anatomical changes. In paralysis after diphtheria and typhus we do not find croupous inflammation, or typhous new formation, in the degenerated nerves. So, in chronic infectious disease, as, for example, in tuberculosis, there may be peripheral nerve degeneration.

Just as ataxy may be a nervous disease, or sequel consecutive to diphtheria, &c., so may tabes be a nervous disease, or sequel consecutive to syphilis. Possibly a persistent chronic slight intoxication by a chemical poison, occurring in the once-infected organism, may induce gradual atrophy in definite lines of nerve fibres.

And, continues Strümpell, the same holds good of general paralysis as of tabes; nearly related, they are in a certain measure, only different localisations of the same morbid process.

As to the above subject, some accept the general principle that syphilis is very important in the production of tabes; others support these views more or less, and either indirectly or directly. For example, concerning the alleged comparative non-effect of anti-luetic treatment, as ordinarily used, in tabes, it has been held by Fürstner that the failure to cure grey degeneration of the posterior columns by anti-luetic treatment, is paralleled by the

[1] Versammlung deutscher Naturforscher und Aerzte zu Heidelberg, Sept., 1889.

fact that diffuse diseases of the central nervous system of specific character are usually not ameliorated by anti-luetic treatment.

On the other hand, objections have been made. Thus, Prof. Schultze, of Bonn, demurs to Strümpell's analogy between tabes and diphtheritic paralysis, for the latter, like ergotin-tabes, is distinguished from genuine tabes, inasmuch as they are regressive, but tabes progressive. In syphilis, there must be a persistent formation of its toxine, against which anti-luetic medication is powerless. Lead-paralysis would be a better analogue of syphilitic tabes than post-diphtheritic paralysis is; for in spite of cessation of the ingestion of lead, the disease often advances.

And as regards the assertion that tabes and general paralysis are so closely akin, or that they are even in a measure only different localisations of the same morbid process, it has been urged by Mendel that, although syphilis is one of the most important predisposing causes of tabes, yet tabes and general paralysis are not to be taken as being related in the way just mentioned; very few general paralytics having grey degeneration of the posterior columns; about 18 or 20 per cent. having loss of knee-jerks; only a very small number, proportionally, of true tabetics becoming general paralytics.

Tuczek affirmed that posterior column changes in general paralysis, do not always (he might have said, do not often) affect the posterior root zones. Lateral column changes mostly co-exist.

H. Schüle's experience is, that so-called classical general paralysis is not so often or so certainly in connection with syphilis as tabes is.

That the connection between syphilis and tabes is much more simple than that between syphilis and general paralysis is held by Fürstner. Posterior columnar disease, alone, is not frequent in general paralysis; more frequent is that of posterior and lateral, or of lateral columns only. In syphilitic general paralysis the complicating spinal cord disease does not always invade the posterior columns.

Recurring to the asserted existence of a close kinship of tabes to general paralysis, or that they are different localisations of the same morbid process, I think we must bear in mind that (as compared with tabes) syphilitic general paralysis is an earlier manifestation of syphilis; is of a more active type; has indeed, usually, irritative inflammatory, exudative and degenerative elements—new formation, destruction, and degeneration, often

simultaneously occupying the tableau—is less chronic and slow than tabes, often, indeed, is subacute; is a primary condition (as a rule) even partly in its degeneration. Whereas, in tabes, the grey degeneration is somewhat analogous to, or partly identical with, secondary Wallerian degeneration (degeneration of nerves consecutive to various lesions, injuries, &c.). But probably it is more precisely akin to peripheral neural degeneration or neuritis, from the toxines of infectious diseases, or from chemical poisons —in a general word, from toxæmic states, including infections, dietetic and chemical poisons, ptomaines, leucomaines; but herein it shows close kinship with general paralysis.

Therefore I think that tabes may be accepted as at least mainly of toxæmic origin, whether from:

Auto-intoxications, altered metabolism; or from

Chronic infections; as syphilis, tuberculosis, leprosy; or from

Acute infections, possibly; (such as diphtheria, typhus, enteric fever, influenza, variola); or from

Ergot—lead—Hg.?—I.?—CO.?

Lithæmia, gouty and rheumatic blood-states generally, diabetes, anæmia perniciosa. (See also auto-intoxications, above.)

In a preceding part of this article, I have mentioned the assertion of some that the lesion of true tabes is only deuteropathic, is only a secondary, a consecutive change in the neuropathic sense. Undoubtedly, in many examples it is a secondary consecutive lesion in the neuropathic sense, and, in such cases, often consecutive to syphilitic disease, sometimes to other ætiological factors. For instance, syphilitic meningitis or gummatous infiltration, or gumma, may give rise to an ascending secondary degeneration or sclerosis; and so of other conditions, such as implication of the spinal ganglia on the posterior nerve roots in some morbid process, however set up; or from peripheral sensory-nerve neuritis or degeneration invading the spinal ganglia, whence, possibly, the morbid process may restart to the cord by way of the posterior nerve roots.

But in many cases of tabes there are not—or not such marked—indications of preceding pachy- or lepto-meningitis, myelitis, gumma, or implication of spinal ganglia or peripheral nerves, or disease of nervous sensory apparatus in skin and muscles—as to explain the posterior nerve root and posterior column change, satisfactorily, as a secondary neuropathic lesion. And the only explanation that commends itself to me, of the tabic lesions occurring primarily, is that there is a selective action upon and of the nerve fibres primarily affected by and to

poison (toxine, &c.) circulating in the blood chronically, much as occurs in nerves in other toxæmic conditions.

The degenerative tendency effective alone may be determined in its operation by local disease. As examples of this, one author cites that set up by a cold bath or by a severe fall, very soon followed by loss of power, undergoing improvement or recovery, but succeeded by the ataxy of tabes.

An ascending neuritic or degenerative change, arising from the seat of primary infection on the genitals, and passing to the spinal cord, whether set up by a toxine, generated locally at first, or coming as an irritative-degenerative process extending upwards by continuity of tissue, is an alternative possibility, to which there are obvious points of demur. In fact, also, the cord and nerve root alterations would *not be primary* in such case.

G.—*The Connection between Tabetic-Arthopathy and Syphilis.*

Two examples are described by Dr. N. Muchin.[1]

The first, a ship captain, aged 41, had incurred primary syphilis eight years previously to his coming under the reporter's notice, and two years after that infection he felt the first evidences of spinal disease. Anti-luetic treatment partially relieved these, but in less than a year the arthropathy of the left foot made appearance. This was greatly ameliorated, indeed practically cured, by specific treatment. Syphilitic spinal paralysis of Erb's type had become fully developed. Clinically the arthropathy was manifestly of nervous origin; the effect of specific treatment, and the clear traces of syphilis, show its syphilitic nature.

The second patient, a merchant, aged 53, had incurred syphilis eighteen years before coming under the reporter's observation. Treated by inunction (*Hg.*?) he felt no further effects for nearly fourteen years, but then the symptoms of tabes dorsalis began. Two and a-half years later, the arthropathy of the left foot began. This recovered under specific treatment.

Muchin concludes that:—

In subjects of tabes, syphilis of the joints may in no wise differ from Charcot's joint disease.

A causative connection between Charcot's arthropathy and disorders of innervation of the joints may be taken as proved.

Yet, not rarely, there are arthropathies which are not of purely

[1] *Deutsche Zeitschr. für Nervenheilkunde*, Bd. v., S. 255.

nervous, but of mixed, origin; wherein the disorders of innervation only serve as predisposing factors. These impair the nutrition of the joints, and form a particularly favourable ground for the action of external and internal noxious agencies.

The most important of such *external noxæ* are mechanical injuries and chills; of *internal*, the chief are syphilis and "arthritismus."

Synovitis of knee-joint may be a manifestation of hereditary syphilis in the young; giving rise to comparatively moderate or little pain or difficulty in walking, and remediable by anti-luetic treatment.

PART V.

"Syphilitic Epilepsy."

A.

If convulsive seizures come on for the first time in a person above 30 or 40 years of age, and otherwise in good health, the chances, according to Fournier, are eight or nine out of ten that the seizures are of syphilitic origin. So-called "epilepsy" is frequent in cerebral syphilis, the explanation of which lies in the fact that the lesions of the latter so often affect the several meninges, or some of them, and the grey cortex.

In cerebral syphilis of "epileptic form," what are the clinical aspects of the "epileptic" seizures? Fournier, while admitting that the "epilepsy" of (tertiary) syphilis is different in nature as a morbid entity, from true, essential, spontaneous, common, "primitive" epilepsy existing from birth or early youth, holds that, *clinically*, the two are much alike, and more so than has been alleged by some; and that, clinically, "syphilitic epilepsy" is a true epilepsy, and takes the forms of ordinary epileptic *grand mal* and *petit mal*, the former being, of the two, much the more common. For convenience of discussion, will here (under section "A") be followed the lines of his description.

Invasion of "Syphilitic Epilepsy."—In a few cases the "epileptic" phenomena, sometimes found in cerebral syphilis, come on alone. Especially may this occur at the onset, and for a limited space of time. But in the great majority of the cases they are accompanied or preceded by other cerebral symptoms, also of specific origin.

As to the time at which they appear:—Sometimes they are the first manifestations of cerebral syphilis, the earliest symptoms to

appear: sometimes they come on later, and only after other symptoms, due to the same lesions, have been established; and sometimes they only first appear shortly before, or at, the closing scenes of the malady.

Subsequent Evolution of "Syphilitic Epilepsy."

This is summed up in three chief points:—

One is, that at first the bouts of seizures, identical with or analogous to each other, nearly always occur at moderate or considerable intervals; but later on tend more and more to occur at shorter intervals. The attacks are preceded in a minority of instances by various mental or sensory prodromata, especially headache, which in some is fixed and circumscribed; and in a few are preceded immediately by auras.

The second point is, that the epileptic form of cerebral syphilis, at its onset and for a time thereafter, may be unmixed, unassociated with other specifically originated cerebral symptoms, and, very exceptionally indeed, this purely "epileptic" clinical aspect may last a very considerable time, and the case may therefore readily be mistaken for one of ordinary epilepsy.

But, ordinarily, and this is the third point, the convulsive symptoms become subsequently modified by, mingled, associated or complicated with, either the symptoms ranged under the name of *petit mal*, or with cerebral symptoms of some other kind, or with both. For the attacks of "syphilitic epilepsy," at first far more frequently of the convulsive *grand mal* type, may subsequently present seizures of one or more of the various kinds grouped under the *petit mal* type. Or, again, the "epileptic" bouts may become associated with cerebral disorders of some other kind, and thus cease to hold the chief or sole place clinically, becoming only one feature in a more complex clinical aspect. The cerebral disorders, one or more of which may thus come to complicate the "epilepsy," are chiefly of three groups; namely, the cerebral congestive, the mental, and the motor (paresis, paralysis).

In the above, Fournier's view seems to have been open to exception on two points: the one in making epilepsy, in the *adult*, from *acquired* syphilis in its *tertiary* stage, show, at first, in almost all cases, the clinical aspects of true essential epilepsy (general convulsion, loss of consciousness); whereas, it is believed, it more often is a "partial epilepsy" at first; the other (p. 186-7) in making the change from the clinical aspect of "essential" or true epilepsy, to that of "symptomatic" epilepsy (if, and when,

it occurs) to be due only to the adjunction of cerebral symptoms of other orders, whereas the features of the attacks themselves assist in the differential diagnosis. Indeed he, impliedly, in part makes good this omission later on.

How, then, is "syphilitic epilepsy" to be diagnosed from ordinary true epilepsy? Does its present special peculiarities in (1) the mode of appearance and succession of its seizures; or (2) in the symptoms constituting those seizures; or (3) in the conditions and facts existent in the intervals between the bouts or seizures?

(1) The mode of appearance and succession of the seizures is not of avail for this purpose. Nor is nocturnal epilepsy specially syphilitic, as it has been alleged to be.

(2) In the second place, as to distinctions to be drawn from symptoms constituting attacks of "syphilitic epilepsy," there is no pathognomonic sign. Some traits may justly arouse suspicion as to a specific origin; such as, in the convulsion of brain syphilis, the usual absence of the initial cry and the much greater frequency of paralysis immediately following the attack. But under this head,

Much more striking, characteristic and useful for diagnostic purposes, are the partial and conscious characters of some convulsive attacks observed in cerebral syphilis. Here (*a*) only a muscular group, or one limb, or one side of the body, may be convulsed; and (*b*) during the convulsion consciousness is more or less retained.

In some cases this retention of consciousness is so marked that the patient becomes a spectator of his own attack—he is said to assist at it—and from it, being conscious, he may suffer severe pain; in other cases the preservation is less complete, consciousness is clouded, and perhaps the patient's replies are monosyllabic only; in still other examples consciousness is confused, obscured, half obliterated during the convulsive movements, of which last the patient does not take much heed or notice, and as to which he evinces no anxiety, at the time.

Sometimes this partial and conscious "epilepsy" forms the first stage of an attack, of which the second stage consists of a more complete seizure with entire loss of consciousness, the convulsive spasms occurring simultaneously, either remaining local and unilateral, or becoming general.

Fournier made far too little of the immediate sequels to these attacks of so-called partial and conscious epilepsy. The mental defects, following, are more frequent and marked than he de-

scribed; and there is *always* (not merely sometimes, as he allowed) some degree of temporary paresis or paralysis after the attacks.

The partial and conscious character of an "epilepsy" is evidence in favour of, though not proof of, its *symptomatic* nature.

Moreover, the partial "epilepsy" is evidence as to the *seat of the lesions* in which it originates; it is the expression of definite localisations, of lesions affecting the grey substance, or the subjacent white, of the cortical motor zone of the brain.

Therefore, as regards the constituent symptoms of the seizures, "syphilitic epilepsy," in some cases, is distinguishable from the ordinary "essential" form by the attacks of partial convulsion, which go to prove the existence of a *symptomatic* epilepsy.

(3) In the third place, some important diagnostic indications are also to be drawn from the facts relating to the intervals between the convulsive attacks. For, whereas the genuine epileptic, as a rule, is fairly well between the several attacks with their prodromata and post-convulsive phenomena; the subject of syphilitic epilepsy, on the contrary, as a rule, is affected, also, with other orders of cerebral symptoms; such as headache, "congestive" symptoms, sensorial disorders, motor troubles, mental symptoms, aphasia, optic neuritis, &c.

Another important diagnostic element is the mature age at which the first convulsive seizures have occurred.

To resume: there is no pathognomonic sign separating "syphilitic epilepsy" from ordinary epilepsy. Yet in some cases it is distinguished by the characters of "partial epilepsy"; namely, localised or unilateral convulsion with more or less preservation of consciousness during the attack. As a rule, also, it becomes associated, before long, with other cerebral phenomena. And, lastly, it comes on at mature age (in usual examples of acquired syphilis).

But having arrived at the conclusion that a "symptomatic epilepsy" is before us, how do we know that it is symptomatic of specific lesions? This we can only know by (1) a valid history, or indications, of syphilis in the past, or (2) the existence in the present of other clear manifestations of syphilis in various parts of the body; and by (3) the exclusion, diagnostically, of possible effective factors other than syphilis; together with (4) the results of anti-syphilitic treatment.

Sometimes we may not have all of even these few points to help us. For example, called to a patient of whom we know little, our only available guides may be the history of preceding syphilis, and the comparativly late age at which convulsion first occurred.

Recently, Fournier has described "para-syphilitic epilepsy" as maintaining through many years the character of true epilepsy, *i.e.*, not associating itself with other cerebral symptoms; as pertaining to tertiary syphilis, and usually coming from ten to twenty years after infection; as presenting both the major and minor attacks; as uninfluenced by anti-luetic treatment, but improved by bromides; and as probably not conditioned by the recognised specific brain lesions of syphilis, although of syphilitic origin.

B.

In section "B" of this part it is proposed (1) to summarise, and then (2) to comment upon, the recent important article of Prof. Paul Kowalewsky[1] on this subject.

I.

He accepts, and builds his articles upon the basis of, a division of epilepsy into (1) cortical epilepsy, and (2) medullary (or idiopathic) epilepsy. Taking these terms, for the moment, in the sense in which they are used, it is evident that until comparatively recent years the former was included in the description of the latter, was not distinctly separated from it, and, though sometimes described as spasmodic epilepsy, even the description of partial spasmodic epilepsy was not altogether that of "cortical" epilepsy, often now so-called.

"Cortical" epilepsy, so-called, is to a large extent, equivalent to syphilitic epilepsy, especially that of *acquired* syphilis, which readily provokes "cortical" convulsion, and is perhaps the most frequent source thereof. In hereditary syphilis, gummata, &c., may induce "cortical" epilepsy, but in many cases the inheritance is, not of syphilis, at least, not of specific local-lesion-making syphilis, but of a weak, defectively stable and resistive, central nervous system.

The epilepsy may come as a result of syphilitic dyscrasia, perhaps affecting nutritive and circulatory centres.

But it may appear as a partial cortical epilepsy, from a circumscribed cortical lesion or disease. In this latter case the results of anti-luetic treatment may be brilliant, the convulsion may only first appear in middle or later life, and is preceded by an aura in the parts which are left temporarily paralysed after the attack. Sooner or later, symptoms of focal lesion usually

[1] *Berliner Klinische Wochenschrift*, Jan. 22, 1894.

co-exist, but in some cases, the centres at fault in originating convulsion are roused to morbid action indirectly, by incitation from a distance.

Epilepsy in Hereditary Syphilis.

According to Kowalewsky *hereditary* syphilis presents itself as of two kinds. One as degenerated, altered, weakened, metamorphosed syphilis; the syphilitic diathesis, so-called, in which the syphilitic contagium is inherited in its chemical form (so to speak); and so far as convulsion is concerned, its effect usually appears as essential, idiopathic, or medullary epilepsy.

The other presents itself as inherited gummatous lesion, either diffuse or syphilomatous [to which should be added[1] the words—or an actively working virus, tending to produce such] giving rise to cortical epilepsy, usually unilateral, or even very local and limited in range.

Inherited syphilis mostly passes over from parent to fœtus as chemical substance, probably chemically combined (or united) with the other elements of the embryonic cells, or serves in these cells, as an equivalent of another chemical substance. In further embryonic growth, this noxious substance may concentrate strongly in the central nervous system, whereby this last is brought into a pathological state, which may manifest itself in all degrees, from gross naked-eye malformations and developmental defects of the brain, down to chemical and molecular defects and alterations in the brain cells, not demonstrable by our methods of examination: and, clinically, ranging from idiocy to slight neurasthenia; but also affording a basis for the later development of neurosis and psychosis of every kind. Taking up only the slighter forms of change, we see, on the basis of hereditary syphilis, the development of neurasthenia, epilepsy, hysteria, chorea, &c. The brain elements containing the noxious substance, seem as if impotent and unresistive towards hostile influences; this leaving an open way for the easy development of every morbid mental and neurosal state. There is, in fact, here, not a special syphilitic neurosis, but a tendency to nervous disease. Thus, in some cases of hereditary epilepsy, syphilis, like alcohol, acts only as a favouring agency. Here the hereditary syphilitic epilepsy is of the essential, idiopathic, "medullary" kind.

It is possible, says Kowalewsky, that in some such cases the syphilitic contagium poisons those of the nervous elements of the

[1] See Comment No. 5, below.

medulla [oblongata?] which serve as centres and starting point of the epileptic attacks, and that it renders their nerve cells more irritable and less capable of resistance. Here, there is irregular function of nerve cells of epileptic sub-cortical centres, owing to abnormal chemical or molecular structure. Therewith operates the syphilitic contagium, either weakening the usual chemical stability of the nerve cells and calling forth quantitative change, or combining with the cell substance and evoking qualitative alteration.

On the other hand, "cortical" epilepsy of hereditary syphilis comes from circumscribed or diffuse formations in the meninges or brain cortex, or sub-cortical ganglia, the diffuse being chiefly in the vascular system. Hence "cortical" hereditary syphilitic epilepsy may present two sorts: the one, with circumscribed formations strongly pronounced and with the characters of quite partial cortical epilepsy; the other, more extensive, and often united with delayed development and innate malformations, and on which, again, polio-encephalitis or meningitis may supervene.

Consequently the two great forms of hereditary syphilitic epilepsy are sharply separated by the mechanism of their commencement, by their origin, prognosis, and treatment.

And one of them, namely, "medullary" hereditary syphilitic epilepsy is taken as having its source in paternal syphilis, whilst the other of them, namely, the "cortical" kind, is held to take origin in syphilis of the mother.

Epilepsy from Acquired Brain Syphilis.

Epilepsy from acquired brain syphilis has usually (or always) the "cortical" type, but even so the mechanism of the origin of this "cortical epilepsy" presents varieties.

(1) The "cortical" epilepsy may be set up (*i.e.*, indirectly, W. J. M.), by gumma of meninges or cortex; or,

(2) By a diffuse gummy process, more intensely affecting cortical and pial vessels at some points or places than at others; or,

(3) During the absorption of diffuse gummy syphilitic products during vigorous anti-syphilitic treatment, by reason of local irritation at the part undergoing resorption; or

(4) By the irritation produced by the scar and contraction of the brain substance, following disappearance of the absorbed gummata.

In all these there is "cortical epilepsy," but in each having a different signification.

In the first, wherein "epilepsy" is produced through the effects of solitary, or of few, gummata (and we may include here,

also, the similar condition in hereditary syphilis—W. J. M.); if untreated, the attacks are unchecked; they are followed by temporary paresis, and, later on, paralysis, and even perhaps contractures. Under energetic specific treatment, the epilepsy may disappear finally and for good, but not seldom it relapses, the convulsion, paresis, paralysis or contracture return, and treatment is without avail. The gumma probably has become replaced by cicatricial tissue and by a local drawing together of the surrounding nervous substance, constituting a focus of incitement to convulsion, and one very little amenable to medication.

Polio-myelitis, or meningitis cerebralis, may come in such cases, and from a syphilitic change in the blood vessels.

In syphilitic epilepsy of this variety the gummata may be recent, may merely compress nerve elements, may yield readily to specific treatment, or they may be of long standing and productive of destruction and disappearance of nervous elements, or the scars and relics of gummata, and of their destructive action, may alone remain.

The second variety of "cortical epilepsy," in *acquired* syphilis, develops with a gummatous process in the meninges or brain cortex, which is diffuse, but some parts of the cortex or membranes, more than others, are affected by this diffuse gummy process in the blood vessels. Under appropriate treatment this may clear up, and the epilepsy pass away. This variety belongs to the lightest of those of syphilitic epilepsy, says Kowalewsky, and he adds that it is followed by no post-epileptic processes and paralyses. To both of these statements one may take some exception.[1]

The third variety of syphilitic cortical epilepsy, or that which appears during resorption of the products of diffuse gummy syphilis under vigorous treatment, is one in which the convulsive seizures occur a very few times only, and disappear, leaving no traces.

To explain this epilepsy is not easy:—

(*a*) Probably auto-intoxication by augmented products of metamorphosis acts here. One may readily admit the possibility of epileptoid attacks brought about by the formation of certain ptomaines and leucomaines in the organism, particularly in persons with defectively resistive central nervous system and a tendency to epilepsy. This form of epilepsy may rightly be termed *toxic;* often the attacks appear as being idiopathic.

[1] See also commentary No. 6, below.

(*b*) Besides this, the vessel walls, in diffuse gummy cortical and meningeal processes, such as have existed in this form of "epilepsy," are filled with neoplastic elements; thus their tonus is much altered. Whilst, under energetic treatment, the vessel walls are being rapidly freed from these neoplastic elements, the irritability and contractility of the vessels is very inequable, and they are inclined to contract suddenly and unequally; as, also, to be dilated and paralysed, and much more markedly so than in normal states. Thus may attacks of cortical epilepsy easily be occasioned, until the normal tone of the blood vessels is re-established.

One or other or both conjointly of these conditions "(*a*)" and "(*b*)" may thus induce this form of syphilitic epilepsy, until:—

(1) The removal from the organism of the unwonted products of regressive metamorphosis occurs; or until

(2) The re-acquisition of due tone of the vessel walls restores a normal circulatory balance and blood pressure in the part, and frees the patient from epilepsy.

For commentary on several of these points see Nos. 2, 3, and 4, below.

The fourth variety of "cortical" epilepsy in acquired syphilis, or that from cicatricial changes and contraction following gummatous processes in cortex or meninges, has already been sufficiently dealt with, incidentally, in discussing the first variety.

Medullary (*idiopathic*) *epilepsy in acquired syphilis* has not been observed by Kowalewsky himself, and he asserts that it is rare. Here must be reckoned the cases of syphilitic epilepsy occurring during the secondary stage of syphilis. This is in consequence of intense concentration of the syphilitic contagium in the central nervous system, and particularly in the region of the "convulsive centres," says Kowalewsky. But we suggest that probably the toxæmic state, equal everywhere in the blood-mass, is sufficient, the vulnerability of the unstable nervous elements, by it, being the effective relation; and selective affinity, for and by —and not concentration of—the toxic substance, being the real modus.

Whereas this form, in which toxæmia is due to the syphilitic virus or its products (toxines), is termed idiopathic ("medullary"); yet that in which products of regressive metamorphosis of diffuse gummy formations which are rapidly dispersing under treatment, enter the vessels and occasion an analogous condition of toxæmia, is termed "cortical" by Kowalewsky.[1]

[1] For comment on this, see No. 3, below.

Summary.

Thus, in short, may all cases of syphilitic epilepsy be placed in two great groups—congenital, and acquired.

Congenital syphilitic epilepsy has two forms: the idiopathic ("medullary"), which almost always develops by reason of irresistiveness and change of the central nervous system produced by the syphilitic contagium, and therefore is rightly termed dyscrasic epilepsy: and the cortical called forth by inheritance of an organised gummy process.

Acquired syphilis also manifests itself in the forms of cortical and idiopathic ("medullary"); the former under the action of isolated gummata, irregular gummy processes, and scars at the site of former gummy formations; the latter under the influence of infection of the organism by the syphilitic contagium, and by the products of regressive metamorphosis, under vigorous antiluetic treatment.

II.

Commentary.—This writer carries one so pleasantly along that judgment is soothed to rest as if by a mental opiate; but I think issue must be joined on several points.

(1) To begin with, the division of epilepsy into "cortical" and "medullary" is one to which we do not adhere. By "medullary epilepsy" (which he does not define), we take it the author means epilepsy considered as a condition of which the clinical symptoms are determined by disorder starting from the medulla oblongata, according to the theory supported by Brown-Séquard and others more than thirty years ago. Under this term "medullary epilepsy"; which Kowalewsky and others use as contrasted with "cortical epilepsy"; are included the cases of epilepsy *par excellence*, those which were chiefly concerned in the older descriptions of epilepsy, and as an appanage of which the more recently (or more generally) separated epileptiform seizures were considered, and the description of which former these seizures at one time modified by their inclusion therein.

But surely all the chief epilepsies are cortical, *i.e.*, the cortex is essentially engaged in them. One may admit that disorder of *motor* grey matter, anywhere in the cerebro-spinal nervous system, may occasion convulsion, or at least an analogous local spasm—analogous, *i.e.*, to a convulsion, or amounting to a convulsion of slight extent, or simple lowly type. Is not "idiopathic" ("medullary") epilepsy as much an affair of the brain cortex as is the epileptiform seizure indirectly set up by a meningeal or

cortical gumma? Indeed, is not the former *more* of a cortical affair than is the latter, involving, as the former does in *grand mal*, large areas, possibly nearly the whole of the cortex? But be this as it may, we accede to Dr. H. Jackson's view as to the implication of the cortex, and Prof. Horsley's experiments on the simian or canine cortex seem to corroborate it.

If we discard the name "cortical" as being misleading if applied to one group only of epileptic cases, how shall we best express the facts? Shall we confine the term epilepsy to what has been called "essential" or "idiopathic" epilepsy, and call the rest "epileptiform or epileptoid convulsions," or "convulsions" merely; or shall we call the former "epilepsy proper," and the latter "symptomatic epilepsy"? This last might be convenient, but is open to the cavil that all epilepsies are symptomatic; although of what symptomatic we do not always know. Or shall we speak of "functional" and of "organic" epilepsy?

Then again, the symptomatic, or even idiopathic, epilepsy may be aroused by excitation at a distance from the nervous apparatus fundamentally at fault. And an important group of epilepsies (convulsions) is produced by various states of toxæmia, whether these last are engendered by the toxines of infectious disease; by drug and chemical poisons; by noxious substances in food, or the outcome of perverted digestion, altered metabolism, or excretory inadequacy of a positive or of a relative kind.

These last are much more analogous to so-called idiopathic epilepsy, than, *e.g.*, is "epilepsy," so-called, from (*i.e.*, the indirect outcome of) intracranial gumma. And, indeed, it is possible that a recurring toxæmic state, or irregularly periodical cumulative tension, resulting from a form of slight auto-intoxication may be one factor in the production of ordinary idiopathic epilepsy, or of some examples of it. For such special form of auto-intoxication might well be due to a functional innate perversion or defect, inasmuch as even structural congenital peculiarities and deviations are observed in undue degree in many epileptics (as, *e.g.*, the osseous, the cerebral).

In the article under discussion, besides (1) the division into "cortical" and "medullary," several other points have been noticed, incidentally, as affording ground for the expression of a different opinion. Such are the following:—

(2) The self-contradiction between the definition of the third form of cortical epilepsy (p. 79, col. 1), and the description of it in the text (p. 80): *i.e.*, between (*a*) the definition as local

irritation at the site of a rapid absorption of diffuse gummatous products, and (*b*) the description as a toxæmia induced by that rapid disintegration and absorption.

(3) The self-contradiction involved in naming "cortical," and describing under that head, the epilepsy due to absorption of products of rapid regressive metamorphosis of gumma, it being in fact, toxic, and therefore a variety of idiopathic ("medullary") epilepsy according to the nomenclature used in the article itself.

(4) The fact that in the third variety described as of "cortical" epilepsy, "cortical" and "medullary" epilepsy, so-called, intermingle; and thus a cross-division is formed.

(5) The qualification that must be appended to the assertion, made impliedly, that cortical epilepsy, in inherited syphilis, means the inheritance at birth, of ready-formed syphilitic gummy neoplasms, or diffuse formations, in meninges or brain; inasmuch as these may form *after* birth.

(6) The denial, that must be made of the statement that "cortical epilepsy" from a diffuse gummatous process is not followed by any post-epileptic phenomena and paralyses, it being invariably followed by some degree of post-convulsive paralysis or paresis.

C.

We now pass entirely away from the excellent article that has been the occasion of the preceding lines.

In all discussions on epileptiform seizures, and of epilepsy, it is very necessary not to lose sight of the influence of alterations of blood-pressure and blood and lymph circulation in the brain, and the relations of these to the cerebral cell elements.

I knew of a case in an asylum in which a portion of the skull-cap had been lost, the part of the brain underlying that area being covered only by the scalp and the meninges. The irregularly recurring convulsions could always be accurately forecast by the preceding bulge of the scalp over the hole, under the increased pressure, from within, of the intracranial contents.

Incidentally, we have noticed how rare a distinguished observer holds "idiopathic" epilepsy to be in acquired syphilis; such cases, for instance, as have been reported in the secondary stage of syphilis. I think it cannot be so very rare, having seen several examples of it, and published two such.[1] Both were

[1] *Brit. and For. Med. Chir. Review*, Oct., 1876, Case 3 in table, p. 451, and text p. 447, and Case 14 in table, p. 454.

cases of epilepsy coming on during the secondary stage of syphilis. In one, chancre was followed in four to five months by "epileptic" seizure, and this quickly by mania, three months after the onset of which there were amygdaloid inguinal and cervical glands and a mucous patch. In the other, the epileptic attacks preceded insanity by several months (five and seven). At its onset, the insanity was termed, elsewhere, "acute dementia," and there was refusal of food; subsequently there was acute, and later on, chronic, mental excitement. On admission, were a papular rash, large hard inguinal glands, a sallow dingy pallor.

As examples of epileptiform convulsions in intracranial syphilis, may be mentioned cases already published by the present writer, such as one with gummatous inflammation of meninges; invasion and destruction of parts of convolutional surface, partly from blockage of vessels; both diffuse and syphilomatous arteritis, chiefly of larger vessels; secondary softenings. (Necropsy.)

Another, with severe osteocopic cranial pains, pachy-meningitis, and dural gummata. (Necropsy.)

Another with pericranial nodes, and severe osteocopic cranial pain. Diagnosis; basal pachy-meningitis (and meningitis?) chiefly posteriorly, gummatous or simple. (Recovery.)

None of these adults had had convulsions until there were other indications of macroscopic intracranial syphilitic disease.

I think we may divide convulsions (not calling them epilepsies) directly or indirectly of syphilitic causation or origin, into:—

(1) Those arising from specific syphilitic intracranial lesion or morbid process, *i.e.*, cellular hyperplasiæ, diffuse or circumscribed, of inflammatory, or quasi-inflammatory nature, in encephalon, or in meninges, or in arteries, and either of a simple type and tending to sclerous change, or of gummatous type; or those arising from histological sequelæ of these lesions.

(2) Those arising more directly from alterations, of syphilitic origin, in blood circulation and pressure, and in brain pressure.

(3) The toxic, or those from what I term syphilæmia, *i.e.*, syphilitic virus or toxine in the blood in the secondary or later stage; or from toxæmia owing to resorption of the products of rapid katabolism of gummy material.

If it exists a congenital or acquired instability of nervous centres will co-operate with all the above.

(4) Those in persons who have inherited both syphilis and a dyscrasia of syphilitic origin manifested (*inter alia*) in nervous instability.

D.

Finally, a word as to nomenclature.

It does not seem to be scientific to call by the same name "epilepsy," the two very different things, to wit, on the one hand:—

First—The epilepsy proper (as it will be called here), having its close relationships with hysteria, with insanity, and with criminality; being—in persons sprung from a neuropathic stock, and presenting somatic and psychic stigmata of neuropathic degeneration—the outward and visible form of an inward and innate vice of nervous-nutrition—of a great constitutional neurosis—the nervous elements being morbidly unstable, liable to gusty, stormy, quick, mercurial change, culminating in paroxysmal outbursts; persons in whom the epilepsy appears from birth or in early life, or in youth; tends to grow with the growth and strengthen with the strength of its unfortunate victims; and, although often associated with obvious developmental defects and deviations, as in cranial bones and in brain, usually presents no neoplastic or other gross perceptible *primary* encephalic pathological change:—and, on the other hand:—

Second—The convulsions, indirectly occasioned by, let us say, the formation of a syphilitic gumma in the meninges or brain of a person probably healthy, or at least not convulsive, hitherto, although now of mature years; the convulsions coming as an accidental occurrence or episode in physical and mental life, and being traceable in ultimate origin to new organic lesions of a grave kind.

To call these two things by the one name "epilepsy"—because convulsion is a striking, overt, dramatic feature in both—seems to me, much as if one clubbed together under the same name—and in consequence of the mental defect in each—on the one hand, the imbecile; and, on the other, the adult whose mind fails because a crowbar has been blown through his head and brain.

PART VI.

Syphilitic Neuralgia.

Syphilitic neuralgiæ have recently been the subject of some clinical lectures by Professor Obolensky.[1]

The first case was that of a male, aged 36, with an old syphilitic history; and some remaining lymphatic gland enlarge-

[1] *Berliner Klinische Wochenschrift*, Feb. 12 and 19, and April 16, 1894.

ment. The pain had first come on nearly five years previously, was worse at night, affected the region of the second, third and fourth intercostal spaces on both sides, was more severe on one side than on the other, and presented the painful points of Valleix at the usual situations.

That the skin is not the part affected is shown by the normal condition of all forms of its sensibility; nor does rough manipulation of the skin increase the pain.

Nor is the muscular tissue affected, because no rheumatic condition, or causative factor of such, exists; and the pain; instead of increasing directly as the pressure increases, as is the case in rheumatism; is increased by slight or moderate pressure, mitigated by strong pressure.

The painful points and their situation, the increase of pain under slight pressure, with its mitigation under strong pressure, the paroxysmal occurrence of the pain, which in the interval remits or ceases—indicate intercostal neuralgia.

The signs of pleurisy are absent.

Neuritis has not existed, for there is no reaction of degeneration on electrical examination, no wasting of muscles supplied by the affected nerves, and no disorder of cutaneous sensibility.

Neuralgia may be produced, as a secondary disease, by mechanical pressure or irritation, as, for example, by tumour; the cause arousing it may be in brain, cord, meninges, or in an inflammatory or other affection of some neighbouring organ or part; or the neuralgia may be reflexly roused from or by, some morbid distant part or organ; or be rheumatic; or follow general nutritive disorder, as, *e.g.*, in the dyscrasia of malaria or of syphilis; or in disorders of hæmato-poietic organs, *e.g.*, chlorosis or leukæmia; or in hysteria, neurasthenia, &c.

Excluding most of the above, for this case, as not supported by examination of the patient, the question whether the neuralgia is malarial comes to the front. With regard to this, besides the general features of neuralgia, above mentioned, the malarial forms present attacks at different times by day or night, and are not fixed, not limited to night time; feelings of chills and heats, and slight rises of body temperature, are apt to occur; intermittent fever has, perhaps, preceded, the patient lives, or has lived in an aguish district. Anti-malarial treatment (quinine and arsenic) assists diagnosis and cures. So, also, does a change of home to a non-malarial district.

Syphilitic neuralgia is distinguished by:—

(1) The history of syphilis.

(2) The existence of traces of past, or evidences of present, syphilis.

(3) Increase of pain at night, its occurrence in paroxysmal attacks at night, the osteocopic character of the pain.

(4) The bilateral disposition of the pain, other forms of neuralgia being (usually) unilateral.

(5) The absence of electrical reaction of degeneration, not-

withstanding the protracted duration of the neuralgia (years perhaps).

(6) The apparently groundless appearance of the pain.

(7) The absence of other causative factors.

Treatment.—By subcutaneous injections of calomel (two only, and at a week's interval, and of about 2 grains each); thermo-cautery over the affected parts; hot baths; galvanisation.

Result.—Recovery, and attributed to the calomel injections.

The second case is that of a man, aged 43 years, who has neuralgic pains in the seven upper intercostal spaces on both sides, but worse on one side than on the other. The sensibility of skin and the muscles are normal. There is no reaction of degeneration, no pleurisy, or vertebral or costal disease. The patient is anæmic, of ill-developed lax musculature. Thickenings, tender on pressure, exist on the frontal eminences and over the nuchal bones, and are painful at night. At the junction of the first and second ribs with the sternum are similar thickenings, tender on pressure.

Left cardiac hypertrophy, double aortic murmur, and aneurysm of the aortic arch are present, and the radial and temporal arteries are sclerosed, harder and more rigid than normal. But the aneurysm is not so situated as to be able to occasion the neuralgia present. Aneurysm of the aortic arch may give rise to a form of neuralgia, or angina pectoris; aneurysm of the descending thoracic aorta may, by pressure, cause intercostal neuralgia, and weakening and retardation of the pulse in the abdominal aorta. Usually, the neuralgia with aortic aneurysm is unilateral.

The patient denies syphilis. Twenty years married, he has five children, whom he states to be healthy, but his wife has twice aborted, and six children have died in infancy of "spasms."

But the diagnosis of syphilis rests on the bone conditions, and tenderness, swelling and pain, already mentioned, including their particular site; on the nocturnal, paroxysmal character of the nerve-pain in head, chest and legs; the enlargement of lymphatic glands in nuchal, axillary, cubital, and inguinal regions; the nocturnal osteocopic leg-pains without periostitis; the long duration; the "groundless" bilateral neuralgia; and the absence of reaction of degeneration.

To syphilis, also, he inclined to attribute the disease of aortic arch and aortic valves, the insufficiency of which explained the vertigo, dyspnœa, and aortic pulsation.

Treatment.—Calomel hypodermically, anti-neuralgic galvanisation, liq. arsenicalis, tr. gelsem. sempervir.

Result.—Rapid recovery from the neuralgia.

The third case, that of a male, aged 43 years, began eighteen years ago with pains in the region of the left kidney, radiating to the scrobiculus cordis and to the breech. The pains began without apparent cause, at first were not very severe, came on

only at intervals of about a month or more, and lasted from twenty to thirty minutes. In the course of several years, they gradually increased in frequency, duration, severity, and extent; so that at last they came on every night, remained for about eleven hours, and, except during short remissions, were intolerably severe; they also took a wider range over left of abdomen and to shoulder and scapula. They ceased suddenly each morning. The night urine, passed during the attacks of pain, was from three to four times the amount of the day urine; it was acid, very pale, its sp. gr. 1008; it was free from abnormal constituents. Insomnia, of course, occurred at night during these pains.

The inguinal glands are enlarged and hard, and those of the neck and axillæ are enlarged. He is married, and has three children: they, and his wife, who has aborted twice, are healthy. At the age of 20, he incurred a hard chancre, followed by enlargement of lymphatic glands and a rash. (*Hg.*)

Vomiting, &c., often occur at the close of, or during, the attacks.

Is the case one of renal colic? Against this diagnosis, are the absence of ureteral pains, or of pains radiating into testicle, penis, or thigh; the absence of any hæmaturia, or of the passage of any calculi or gravel, during the long space of time presenting so many and such severe attacks of pain: the absence of pain in, or tenderness on pressure over, the kidney, and of abnormal urinary deposits. Movement, straining exertion, or an incommodious vehicle, do *not* affect or bring on the pains, as they are apt to do with renal calculus; and, unlike what occurs in this last, the pain is straitly confined to the night-time.

The long duration of the shooting pains, without any other indications as of tabes, shows that the attacks are not the renal crises of that disease.

Nor is the case one of diabetes insipidus; the over-secretion during the night of pain is fairly well balanced by the under-secretion during the day of freedom; and there is no thirst. There are no symptoms of interstitial nephritis.

The pains are neuralgic, coming on without apparent cause, and ceasing suddenly, sometimes with vomiting.

The pains are partly in the same places as those of renal colic, and during them the function of the kidneys is disordered. Therefore the source of the neuralgia appears to be the nerves which innervate the kidneys, and both the sensory and those which regulate the vascular tonus are affected. There is neuralgia of the splanchnic nerve, accounting for the pain; and, on the vasomotor side, dilatation of the renal blood vessels and increased watery secretion of urine, owing to periodical changes in the splanchnic nerve, or, perhaps, renal plexus.

The neuralgia is not symptomatic of a cerebral, or spinal, or bony disease. Nor is there the slightest evidence that it is reflexly-produced pain; nor is there any indication of neuritis of ordinary full-blown type.

That the origin of the neuralgia is not malarial is concluded on grounds similar to those for the same conclusion in a case stated in summary above. That the neuralgia is of syphilitic origin is concluded from the nocturnal character of the pain, the history of lues, and the enlarged lympathic glands.

Rapid recovery under (as he refused Hg.) K.I., Na.Br. Gelsem. Semp., As., thermo-cautery over spine, galvanisation.

(K. I. fails in some cases.)

The diagnosis of syphilitic neuralgia rests on :—

(1) The nocturnal occurrence of the neuralgic attacks.

(2) If intercostal the neuralgia is bilateral.

(3) The absence of the reaction of degeneration and atrophy, notwithstanding the long duration of the neuralgia ; such signs indicating neuritis rather than neuralgia.

(4) The appearance of the pain without apparent occasion.

(5) The history or indications of past or present syphilis.

In contrast with the above, may be mentioned two cases of irritative peripheral syphilitic neuritis reported by S. Ehrmann.[1]

One was a coachman, aged 38. Paræsthesia in region supplied by the left ulnar nerve occurred eight and a-half months after syphilitic infection. The interossei and other small hand-muscles were extremely atrophic. Hyperæsthesia affected the areas of distribution of certain nerve-branches in forearm and hand. Galvanic irritability lessened ; no reaction of degeneration. The ulnar nerve was somewhat thickened, and in its entire length was painful on contact. Anti-luetic treatment succeeded.

The other was a merchant, aged 32. Pains in left thigh a year after infection. The peroneal nerve distinctly thickened, tender, painful. Hyperæsthesia in the district of the crural nerve ; hyperalgesia in the cutaneous areas supplied by the peroneal nerve. Slight bladder and rectum disorders. Vastus muscles very much atrophied.

[1] *Wiener Med. Wochenschrift*, No. 33, 1893.

Critical Digest.

SOME RECENT PSYCHOPHYSICAL LITERATURE.

Professor Kuelpe[1] has given us a text-book of experimental psychology; and Professor Sanford[2] is writing a Laboratory Course. M. Binet[3] now steps into the field with a popular Introduction to the science, in the compilation of which he has been assisted by MM. Philippe, Courtier and v. Henri. If the first two of these works are good—so good that psychologists may be excused for feeling a little self-congratulatory for the moment—it must be confessed that the last is somewhat disappointing. Opening with a full account of the Laboratory of the Sorbonne, followed by a sketch of the Leipsic Institute, it devotes seven brief and incomplete chapters to the discussion of psychological method and achievement. Two only of these appear to have been written with any degree of enthusiasm—those on movement and on memory. The former, indeed, is so tantalisingly interesting and lucid, that one cannot but wish there were more of it, and more like it. After these rank the sections on "psychometry" (the word looks better in French than in any other language, but is bad enough even there), and on observational method. The remaining three chapters are entitled psychological method, sensation perception and attention, and ideation.

Besides being unworthy of an author of M. Binet's power and experience, the volume sins in the matter of comparisons which are odious. The writers do not state that experimental psychology is *une science française*, but they never tire of congratulating themselves that they are not as the *expérimentateurs étrangers*. This is certainly to be deprecated in the interest of

[1] "Grundriss der psychologie, auf experimenteller Grundlage dargestellt," Leipzig, 1893.

[2] "A Course in Experimental psychology," advanced sheets, Boston, Mass., 1894.

[3] "Introduction à la psychologie expérimentale," Paris, 1894.

the science: and the danger of a recoil is great—as is curiously illustrated in the present instance. For the great difference between the old psychology (of which Wolff may stand as typical), and the new (of which Wundt was the effective founder) is this, that the former was merely descriptive, while the latter is descriptive and explanatory. The Wolffians—I use the term more broadly than is, perhaps, legitimate—depicted isolated mental facts; described separate complex processes, such as emotion; and furnished none but metaphysical explanations of the course of psychical events. The moderns seek to give psychological theories, that is, to state the psychological conditions of the processes of consciousness; they work not upon a "faculty" here and another there, but upon the total extent of mind, searching for uniformities and principles of correlation. The result is, that the stricter experimentalists—those who define "experiment" in psychology as they would in physiology—tend to work predominantly upon the normal, healthy consciousness, having recourse to the abnormal only for verification or illustration. But it was a result of equal inevitableness for the descriptive school that it should have a keen relish for the abnormal—that it should devote the time it did to the investigation of "genius," phenomenal memories, and so forth. And lo! I have before me, for notice, three other books of M. Binet's, and they are entitled "Les altérations de la personnalité,"[1] "Psychologie des grands calculateurs et joueurs d'échecs,"[2] and "On Double Consciousness."[3] Here is a glass house, in very truth.

Of course, I do not mean that the methods of this author are the methods of the seventeenth century. His problems are all conceived in the spirit of modern science. Nor is it anything more than an accident that the volumes under review deal with abnormalities. M. Binet and his pupils have published too many experiments upon memory, tactile localisation, &c., for there to be any valid ground for the counter-criticism that the study of the unhealthy or the out-of-the-way is characteristically French, or that there is any radical difference between the methodology of experimental psychology in France and in Germany and America.

As regards the general question, however, there can be no doubt, as Kuelpe says, that the students of mental pathology are far more indebted to the students of mental physiology than are these last to the former. The very phrase "double consciousness" is indicative. It presupposes, obviously, that in employing the

[1] Paris, 1892. [2] Paris, 1894. [3] Chicago, Ill., 1894 (Trs.)

term "consciousness" we are speaking in terms of the known; by reducing the given problem to a "conscious" formulation, we are helping matters on towards its solution. In other words, the pathologist is appealing to physiology for explanatory analogies. Unfortunately the appeal has an *ad captandum* ring. The phrase seems to have gained an established place in psychological nomenclature: it has been brought into vogue by such men as Taine, Janet, Dessoir, Moll. And yet, as Wundt has pointed out, it is nothing else than a rehabilitation of the old beliefs in demoniacal possession, in second sight, in supernatural illumination. Of necessity, its connotation has been modernised; the "second consciousness" has fallen from its high estate; from being the more important—a special endowment or affliction—it has become the less important, and we talk, in would-be scientific terms, of the "subliminal" mind, or with Myers of by-paths, stray tendencies of conscious evolution. Are we any the nearer to a real explanation of the phenomena?

This is all very well, it may be said: but has the "strictly scientific" school any "real" explanation to offer in place of the one it condemns? Now such a question cannot but put the questioned at a disadvantage. For plainly, people can understand and remember the word "phlogiston," but the proof that heat is a mode of motion requires time and patience for its assimilation. So the "hypothesis" of a complex self is readily grasped, and has something fascinatingly uncanny about it; whereas the grasping of the other type of theory means a fairly detailed study of certain portions (not the most easy) of physiology and psychology. If the reader will take the trouble to work through the writings of Moll,[1] Bernheim[2] and Wundt,[3] he will have some idea of the actual difficulty of the problem, and of the reasonings put forward towards its solution. As regards the essay more especially under discussion, it is noteworthy that even M. Courtier[4] will not go the whole way with M. Binet. But where is the line to be drawn?

The volume mentioned above in the second place is more valuable. It contains exhaustive studies of the mentality of MM. Inaudi—a professional "calculator" of the exclusively auditory type—and Diamandi, a visual calculator. It also publishes the

[1] "Der Hypnotismus," Berlin, 1890, second edition.

[2] "Die Suggestion une ihre Heilwirkung" (Trs.), Leipzig und Wien, 1888.

[3] "Hypnotismus und Suggestion," Philosophische Studien, 1892. Also separately.

[4] *The Psychological Review*, i., p. 194.

results of an *enquête* upon blindfold chessplaying, the outcome of which is, that the players possess an unusual power of sustained concentration of the attention, a highly developed faculty of visualisation, and a reliable recapitulatory memory. Both sets of facts are interesting: they are material upon which the psychophysicist of James' "unborable" category can work by way of further analysis.

The third work—a volume in the International Scientific Series, in which the "Animal Magnetism" of the writer and M. Féré had already appeared—contains a popular exposition of the facts of changed personality; dealing with "successive personalities," "coexistent personalities," and "alterations in personality induced by suggestion." It is the author's purpose to avoid theory: "je retiendrai seulement les expériences qui se repétent entre toutes les mains, et qui donnent toujours la même conclusion, quelle que soit la fin cherchée." He has been conscientious in adherence to this plan; and although his own views appear clearly enough when looked for, and the hypothesis of divided consciousness is at times strained to breaking, we have in the book an assemblage of scattered observations which it was well worth while to bring together, and which are stated fully and without prejudice. The title and to some extent the contents of the treatise naturally recall M. Ribot's "Diseases of Personality."[1]

Before taking leave of M. Binet I may, perhaps, call attention to the second edition of his "Études de psychologie expérimentale."[2] The volume contains four papers: on fetishism in love, a pathological study based on the work of Charcot and Magnan but embodying much new material; on the psychic life of micro-organisms; on the intensity of mental images; and on the hypnotic problem, together with a note on hysteric writing. The second article presents a number of valuable biological observations, illustrated by diagrams for the most part after Professor Balbiani. As regards the psychology, that is as it must be, analogical and speculative. The author, though he ascribes to the Infusoria a faculty of choice, is not carried to extremes by falling into the language fallacy, as, *e.g.*, Verworn[3] has been. This "choice," for instance, is "un des faits es plus obscurs. . . . quant à l'interpréter, nous en sommes incapables." But is not the term, on that account, a dangerous one to employ? The third is a discussion based upon hypnotic results, but con-

[1] Chicago, Ill., 1894 (Trs.); Paris, 1889, 3rd ed.
[2] "Le fétichisme dans l'amour, &c.," Paris, 1891.
[3] "Psychophysiologische Protistenstudien," 1889.

tains remarks on the relation of logic to psychology, of the association of ideas to belief, which bring the author into closer connection with experimental psychology in the German sense. Lastly, the fourth paper tells us in succinct terms of the gravamen of the Nancy school against that of the Saltpêtrière.

Next upon my list stand two volumes dealing with memory.[1] Neither of them can at all compare in importance with the work of Ebbinghaus,[2] or with that of Mueller and Schumann[3] which arose out of it. Professor van Biervliet's study seems to have been intended as a brief popular introduction to psychology in general, and as such it may serve a good purpose. It has hardly anything of the nature of a true monograph. The author explains the fact of retention by a combination of the Maudsley-Ribot-Delbœuf and Wundt theories, coining the word *trace-disposition* to express his view. He finds three kinds of memory: that of fixation—the more frequent the stimulus, the readier the functioning in response to it; that of reproduction, which is regarded as entirely passive, and as governed by the association "laws" of contiguity and succession; and that of identification, a "faculté de l'âme," not (like the other two) common to mind and matter. There follow short paragraphs on the physiology, pathology and hygiene of memory. But the book, comparatively superficial as it is, will at least be helpful to the beginner; which is more than can be said for that of M. Verneuil. This is a rambling, anecdotal treatise, unchaptered and unindexed, dealing with such questions as the reality of innate ideas, physiognomy, the nature of the mind, cerebral retention, inattention as a cause of weakness of memory, "corps éthéréens," the relation of imagination to memory, memory in animals, heredity, a universal natural science, &c. What the author exactly means by his references to the "cells in the grey substance of the immense brain of God" I have not been able to discover. His general drift seems to be—though I may have misunderstood him—that the functions of memory depend upon nervous functionings, and that this dependency need imply neither Materialism nor Spiritualism.

Feeling and emotion have been very prominently upon the psychological carpet of late. The James-Lange theory is under

[1] "La mémoire," J. J. van Biervliet. Gand & Paris, 1893. "La mémoire au point de vue physiologique, psychologique et anatomique," H. Verneuil. Paris, 1888.

[2] "Das Gedaechtniss," Leipzig, 1885.

[3] "Experimentelle Beiträge zur Untersuchung des Gedaechtnisses," in the *Zeitschrift für Psychologie*, &c., 1893. Also separately.

discussion in the technical journals; experimental investigations into the uniformities of the sense feelings have been undertaken in more than one laboratory; and the publication of Lehmann's "Hauptgesetze des menschlichen Gefuehlslebens"[1] marked a distinct advance in affective psychology. Four works call for notice in the present connection: and firstly Féré's "La pathologie des émotions,"[2] and Bourdon's "L'expression des émotions et des tendances dans le language."[3] The former is a ponderous tome of 600 pages, *de omnibus rebus et quibusdam aliis*, written with no petty sense of restriction to the subject indicated in its title. As we are to deal with man, the author seems to say, let us put together everything that we know of him that can possibly be of any use to us. Let us see how he is influenced by atmosphere and temperature, by sounds and sunlight; and mark the effects of physical exercise or the lack of it. We shall get to the emotions in due time. As we do, at about the 170th page.

M. Féré does not appear to have any psychological theory of the emotion. Whether it is an ultimate psychical process, or a fusion of the pleasure-pain element with ideational masses, or a complex of organic sensations, he does not tell us. His chief principle of classification is that of the sthenic (*vives, excitantes, violentes, intenses*) and asthenic (*dépressives, penibles*) types. But this is crossed by the joy-sorrow division (*agréables, tristes*), by a division based upon cause (*provoquées, suggerées*), and by yet another based upon the objective reference of the emotional state (*morales, maternelles*). The reader who comes to the work for results of psychological analysis, therefore, will lay it down disappointed.

Not that the book does not contain matter enough. It treats, in an encyclopædic way, of the pathological and curative effects of emotion, of the results of excitement and fatigue, of various psychopathic manifestations, of the forms and conditions of morbid emotivity, and of its diagnosis, treatment and prophylaxy. An interesting chapter, which has come into the book for no particular reason that is apparent, deals with the physical signs of hallucination. The pages devoted to attention bring together a number of facts which are often overlooked in the normal psychologies. But it is curious to read such a statement as this, in face of the general pyschological consensus that attentional phenomena are primarily inhibitory in nature:—"L'exaltation générale de l'activité motrice . . . semble demontrer que c'est

[1] Leipzig, 1892. [2] Paris, 1892. [3] Paris, 1892.

à tort que l'on fait intervenir l'hypothèse de l'action de l'arrêt, de l'inhibition, dans la physiologie de l'attention." And it is also curious to find attention figuring in a catalogue of emotions (pp. 115, 207, 208); although many writers emphasise the characteristic feeling which accompanies attention; and it is not unusual to have expectation (expectant attention) classed as an emotion in the strict sense.

The fact that the volume is so altogether unreviewable tempts one to linger upon its by-subjects. A good deal is said about reaction times, dynamography, ergography and the relation of sensation to movement in general, a favourite topic, of course, with the author.[1] I notice that he takes the side of Wallace in regard to the sexual selection question: "Ces mouvements (des oiseaux mâles) ne sont que des décharges involuntaires du systeme nerveux arrivé a un degré extrême de tension" (p. 213). He has a novel theory of the action of the pneumogastric in syncope and death consequent on emotion. As regards the "seat" of emotion, "Le grand sympathique parait être son organe peripherique," "L'emotion est essentiellement un phenomene reflexe generalisé, dont la voie centrifuge est principalement le systeme grand sympathique." Any kind of cortical localisation is impossible. Emotional expression is mediated "par des mouvements des organes dont l'innervation est le plus souvent en jeu, et en particulier les organes circulatoires." Secondary factors are the length of a nerve, its volume, and the vascularity of the regions it traverses. If we compare this mechanical view with the explanations given by Darwin and Wundt we see that it partly coincides with the first (association of serviceable habits) and third (action due to the constitution of the nervous system) principles of the former author, and also with the first (direct modification of innervation) and third (connection of movement with sensation) of the latter. It seems too simple to cover all the facts.

Given a large assortment of newspaper cuttings and of records of cases from medical practice, a good index to medical periodical literature, and a copious stock of patience, and it would be possible for anyone of ordinary intelligence to fill 600 octavo pages on the variety of topics that M. Féré covers. But the result would not be M. Féré's book. The author writes with a kind of suppressed enthusiasm, a settled conviction that all his by-the-ways and first-of-alls will of necessity interest the reader; he

[1] *Cf.* his "Sensation et mouvement," Paris, 1887.

has humanity on the dissecting table, and means to know all about it. It is only fair to say, too, that he is very careful in his use of authorities. When once one has learned one's way about the book, it is valuable on many counts.

I will just mention here, in passing, a much more typically French work—better in style and better in arrangement—upon a special pathological subject, "La contagion du meurtre," by M. Aubry.[1]

M. Bourdon's book brings psychophysics into relation with philology. Emotion is, for him, "un fonctionnement anormal passager d'un organisme animal": a statement which reminds the reader of Ribot's characterisation of attention. It is the mental resultant or correlate of a total "organic" state; and as such, is no more to be reckoned to sensibility than it is to volition.[2] This view is paralleled by the author with Professor James' theory. But it plainly differs a good deal from that, for it is sensations ("organic" sensations) that Professor James insists on, both in his original exposition[3] and in his recent *apologia* in the *Psychological Review*.[4] The word "organic" is, of course, equivocal. In type, however, the two conceptions are similar. A "tendency" for M. Bourdon is "l'habitude d'une sensation." Both terms, then, are used in wide senses, such psychoses as hunger, sex-instincts, the reaction upon a colour, being regarded as emotions, along with anger, fear, &c.

The list of the special laws of emotion is incomplete, but suggestive. It should be compared with Lehmann's sections on the same subject. The criticism of the explanations of emotional expression given by Darwin and Wundt is very acute, and must, I think, be taken account of by future psychologies. I cannot well summarise the writer on either point; the questions at issue are questions of detail.

We now come to the main topic of the work—the spoken word. This is considered under the headings of pitch, intensity, form or quality, duration, pause (*arrêt ou silence*); and then each of these elements is dealt with in composition, successions of intensities, &c., successions of words, syllables, &c., cohesion and dissociation. A final section on special problems treats of the useless elements of spoken language, of linguistic categories, of poetry and of writing.

[1] Second edition, Paris, 1892.

[2] Pp. 3, 17. Cf., however, p. 9: "l'émotion, comme la sensation, dont elle n'est qu'un accroissement. . . ."

[3] "Principles of Psychology," ii., pp. 449, 450.

[4] Vol. i., No. 3.

These chapters are very far from being free of error in philological detail.[1] Such error, however, might be at least partially condoned in presence of a sound methodology. The author's method is the statistical. He analyses laboriously into their elements passages selected under the guidance of certain principles from the works of various authors in different languages. But the method does not lead in his hands to the right *kind* of result. Thus, to take what is perhaps the most striking instance: there are long tables showing the relative frequency of dentals, labials and gutturals in French, German, English, Italian, Spanish, Celtic, Russian, and Hungarian. But these languages, instead of evincing differences of such a sort that they might be correlated with emotional differences, give all of them about the same proportions of usage. The writer, instead of modifying his method, proceeds irrelevantly to evaluate this result, deducing from it (1) a general law of human speech—general, because of the eight languages one is not Indo-European; and (2) referring it to the great tactile sensibility of the tip of the tongue, its motility, and its juxtaposition to the teeth. It need hardly be remarked that the generalisation is over-hasty; and that, even if true, it is not the kind of generalisation wanted in the book.

The author summarises his work in three propositions. (1) The only direct action of emotional intensity upon ordinary speech is dynamogenic. Moderate emotion increases the force of respiration, and with it the intensity of the spoken word; violent emotion weakens or inhibits the voice. (2) Language is primarily a phenomenon of imitation. (3) The degree of organisation and of stability of linguistic combinations (*unités*) is, other things equal, inversely proportional to the number of elementary phenomena included in them. Not only does M. Bourdon here do his chapters injustice; he has again, it would appear, lost the point of them. Is the influence of emotional intensity, *e.g.*, permanently embodied in the utterance of particular peoples? We are not told.

One lays the volume down with an irritated wish that, containing as it does so many indications of ability, it should not be altogether better. But it is, in any case, well worth reading.

I pass to M. Godfernaux' "Feeling and Thought."[2] The former term covers the phenomena of the affective life, the origin of which is subjective or internal; the latter those of the representative,

[1] This has been pointed out by the anonymous reviewer in *Mind*, new series, vol. ii., p. 403.

[2] "Le Sentiment et la Pensée," *Thèse de Doctorat*, Paris, 1894.

whose origin is external or objective. The aim of the work is to reinstate the first in their proper place in the structure of mind, in the place from which they have been deposed by current associationist theories.

The author sets out to find the two psychoses in isolation. He discovers a morbid exaggeration of thought in mania and hypochondria. "Dans la manie, la connaissance du monde extérieur, dans l'hypocondrie, les éléments de la notion de notre moi corporel sont peu a peu dissociés jusqu'à la sensation ou l'image simple." In melancholy and ecstacy, on the other hand, feeling is seen in all stages of exaggeration, up to absolute predominance. Finally, in chronic delirium the quantitative relation gives place to a qualitative; we have "une succession définie et constante d'états affectifs differents, correspondant à des modifications definies et constantes de l'association des idées." I do not know how far the correlation of hypochondria with mania will meet with medical approval: the writer's analysis is ingenious. But, that apart, he evidently pushes his instances beyond what they will bear, in his eagerness to exemplify his standpoint.

Passing to the study of the normal consciousness, M. Godfernaux notes the occurrence of affective states of excitement and depression similar in kind to those observed in the abnormal mentality. He further proceeds to show that the affective state is normally *l'agent profond* of the association of ideas. In order to make his view of the interrelation of the two sides of mind more precise, he has recourse to the phenomena of movement, and concludes that "the work by which the vague and indefinite effort of the tendency (*tendance*) becomes definitised in more or less complex groups of motor phenomena, and, ultimately, in very well defined muscular co-ordinations, shows a constant correspondence to the work by which the emotion takes on a concrete and definite form, and creates a definite synthesis of the elements of consciousness, sensations or simple images." Four appendices deal with the evolution and classification of mental derangement, and a fifth records some observations on chronic delirium.

The writer might with advantage have been more exact in his use of terms. "Feeling," he describes as "un phenomène de conscience diffus et vague." It apparently includes affection proper and rudimentary conation. Had the literature been better searched, this vagueness could have been removed. All citations (except one from Horwicz and one from Lourie) are

from French works or translations; thus, Wundt is quoted from the French version of the second edition; Lehmann is not mentioned, although he and M. Godfernaux treat in part of the same topics. At the same time, the book is welcome as indicating a reaction against the exclusively associationist position. The recent articles upon attention and apperception in BRAIN will have made its readers familiar with the fact that there are a large number of psychologists who cannot rest content with a reduction of conscious life to sensational terms. Emotion is, like attention, a process requiring other conscious elements for its satisfactory explanation.

Dr. Irons has been writing in *Mind*[1] recently to show that the emotion is not a complex of elementary mental processes, but itself a fact of mind which is *sui generis*. The ordinary opinion is that emotion is a compound of the affective element (pleasure-pain) with ideational masses, these latter differing in the different emotions. Just as a headache and a toothache differ only sensationally, the sensational substrate of the affection being different and enabling us to localise the aches in forehead and tooth, and are identical in affective quality, the same *qua* pains, *qua* unpleasantnesses, so the emotions are all alike pleasant or painful, all fall within one of these two affective qualities, and differ simply in regard to the trains of ideas, associations, imaginings, &c., which constitute them on the intellectual side. This view does not, of course, as so many authors assert it does, attempt to reduce all concrete pleasures and pains to one type, and all the various emotions to simple pleasure and pain. It says merely, that just as the musical chord has a quality of its own (the piano chord being, *e.g.*, readily distinguishable from the violin clang), but yet is composed in the last resort of a number of simultaneous separate sensations, so the various emotions, qualitatively different as they are in immediate experience, admit of ultimate analysis into one of the two affective qualities, *plus* a particular set of ideas. That is why the emotions cannot be classified; to classify them one would have to classify one's ideas, for any idea can enter into an emotion.

I have dwelt upon this point because M. Danville's little volume on Love[2] is intended to prove that this emotion is irreducible to any mental elements beyond itself. After a somewhat tedious introduction, and criticisms of the views of

[1] New series, vol. iii., pp. 77, ff.

[2] "La Psychologie de l'Amour." Paris, 1894.

literature and philosophy (Bain, Spencer, Mantegazza, von Hartmann, Schopenhauer, Delbœuf, Beaunis, .Pierre Janet, Féré, de Fleury), we come upon the author's definition. Love is a specific emotive entity, consisting in a more or less permanent variation of the affective and mental state of a subject, occasioned by the realisation (due to the fortuitous initiation of a specialised mental process) of an exclusive and conscious systematisation of his sexual instinct upon an individual of the opposite sex. The phenomenon is, as a rule, accompanied by an exaltation of desire. The first clause means that love "est irreductible à tout autre sentiment." I do not suppose that anybody would dispute this. The exaggeration of desire is regarded as a purely secondary, indirect character. The specialised mental process which "préside à l'origine de la passion amoureuse" is treated in a special chapter, entitled "Psychological Theory" of love. Life at first is asexual. Then follow the various stages of sexuality; unselective sexuality, selective sexuality, relatively systematised selection, love. Each of these has correlated with it a special level of intellectual development. The particular intellectual process which initiates love is a conscious perception of what has heretofore been an "unconscious perception," the perception of an image representing the synthetic type of the particular preferences of the individual from the point of view of sex.

A second point discussed in the book is that of the nature of love as normal or pathological. The writer sums up the evidence, as he has done before in shorter compass in the *Revue Philosophique*,[1] and decides for normality. "Il est un argument décisif, criterium de l'utilité, qui demontre que l'opinion [contraire] ne parait pas soutenable."

It is clear that there are weak points in the definition which forms the pivot of the whole discussion, even if the author had not incidentally committed himself to the phrase "une passion aussi complexe que l'est la passion amoureuse." But apart from this the chief interest of the book is that it is a book of tendency. It is popularly written, and makes no claim to furnish anything more than a cursory treatment of its subject.

"Die drei Wege des Denkens,"[2] by Dr. I. Paul, is an essay upon the three possible modes of thought about "things"—the physical, psychological, and metaphysical. The writer is influenced almost exclusively by Schopenhauer and Fechner. He insists

[1] March, 1893. [2] Leipzig, 1891.

very strongly upon the fallacy involved in the concept of "transcendent causality"; the doctrine that the interrelation of mind and body is to be conceived of as causal.

Professor Fano's lecture on the "Physiological Substrate of Thought"[1] is interesting and suggestive. After stating the parallelistic principle in a simple form, he gives his auditors, again very simply, the key to the structure of the nervous system, and gets to his special topic by way of some experiments on *Emys europæa*, made by Francesco Redi in the middle of the seventeenth century. He goes on to present a functional analysis of the tortoise brain, using it to illustrate the fact that all katabolism is normally followed or attended by anabolism. "Ma perchè gli elementi che constituiscono l'organismo continuino a vivere ed a funzionare, occorre che al lavorio distruttivo della molecula organica, che è il sottostrato chimico delle nostre funzioni di movimento, corrisponda un' attività riparatrice per la quale si accumulino di nuovo nella cellula funzionante quelle energie potenziali che essa ha speso col lavoro." Consciousness (will and perception) is correlated with the first phase of this double process; it is a phenomenon of inhibition. "Come le resistenze interposte in un circuito elettrico possono esprimersi sotto forma di luce e di calore, cosi la coscienza che accompagna i movimenti provocati dal cervello e la intelligenza che per essi si rivela, sono il risultato della resistenza presentata dal cervello medio nell' ambito psichico." Its second phase, of organic synthesis, constitutes the physical basis of memory, "il fondamento delle funzioni mnemoniche." There is nothing new in these views, of course; but the lecturer puts them well. Original with the writer, on the other hand, is apparently the following opinion: "Mi sembra si possa concludere che gli stimoli alle attività trofiche di ricostituzione organica consistano in alcuni casi almeno nei prodotti, nelle scorie, nelle ceneri che sono il risultato delle funzioni."

Still lighter in matter and treatment is the same author's "Le funzioni del cuore nei sentimenti."[2] The paper opens with a sketch of the mechanics of heart-beat and a reference to the part played in physiology by rhythmically recurrent processes. It then passes to psychophysics; the surface of the heart is insensitive, the movements of the organ beyond our volitional control. Why, then, do language and popular opinion so universally regard it as the seat of feeling, passion, and emotion? Because of its unique

[1] "Di alcuni fondamenti fisiologici del pensiero," Milano-Torino, 1890. (*Rivista di Filosofia scientifica*, April.)

[2] Trieste, 1893. ("Raccolta di scritti di medicina per côlti profani.")

connection with the brain. This connection is such that, in the first place, the heart is the principal bodily reflector of the mind: "Quasi ogni stimolo che arrivi al cervello dall' esterno, ogni pensiero che vi si svolga, ogni emozione che lo scuota può essere trasmessa per la via dei nervi cardiaci al cuore ed esprimersi nei suoi palpiti." Heart is to brain as actor to dramatic author. In the second place, we have the heart reflecting physiologically caused emotions; it may be by way of the nervous system again, or it may be under the direct influence of the quality of the blood. The tissues are composed of numberless minute organisms, cells: "Il cuore per le sue disposizioni anatomiche e funzionali può perfettamente risentire e riprodurre le voci misteriose" of these humble creatures. This recalls Delbœuf's theory of love—that the sovereign wills of an ovule and a spermatozoön impel the youth and the maiden to seek each other's company. The lecture is not so serious a piece of work as that just noticed; the psychology in it is very incomplete.

Similarly defective in general psychological grasp is Professor Brugia's "Pensiero e senso organico."[1] This is the greater pity as the writer's use of method in weighing evidence seems more than averagely good. He begins by emphasising the solidarity of the organism: though the viscera are controlled by special centres, and act and react on one another with a certain degree of independence, yet in the last resort the highest-level centres are the appeal, and they have the final responsibility. Mind is "una forza insita nel cervello," which is at the same time "l'organo della recettività cenestesica." Or again, mind is "la sintesi di tutti i fenomeni somatici." To the question of the nervous connections between viscera and brain the author has no answer of his own; he carefully reviews the evidence, and concludes that we have certain knowledge only of the points of arrival and departure. He emphasises the psychological value of cœnæsthesia: "è sopratutto l'oscura coscienza organica che dà colorito alle impressioni e alle idee, che . . . determina il modo d'essere e di reagire delle cellule nervose corticali." The psycho-organic co-ordination becomes less complete as mentality develops. The doctrine of temperaments is set forth under the rubric "influence of the cerebral cortex on the organic constitution;" and then follows an important chapter on visceral disturbances in the ætiology of psychoses. Dr. Brugia holds what may be called a waste product theory ("una possibile origine

[1] "Saggio di psico-patologia della cenestesi," Aversa, 1893.

tossicoemica," "un ricorrente processo di autointossicazione della corteccia," are characteristic phrases). It is curious to find this hypothesis based upon the analogy from sleep. Surely a waste-product theory of sleep, whether the product favoured be lactic acid or carbonic acid, cannot avoid the necessity of a supplementary hypothesis, introducing the play of an inhibitory mechanism. And a bolstered theory is never quite satisfactory. As a set-off to this slip I cite a remark which is too good to be overlooked: "Per ciò che concerne là delimitazione delle influenze somatiche che agirebbero 'per contraccolpo' sul cervello," writes the author, "è chiaro che . . . un profondo studio di molte psicosi che hanno apparamento forma reflessa potra scoprire qualche nuovo fattore genetico, insieme a una condizione che, per essere la piu conspicua, può sembrare la sola operativa." The only other general chapter deals with the organic evolution of instinct. The views of Darwin, Spencer and Romanes are discussed with some acuteness; but there is no indication that the writer has put the question as to what an instinct *is* psychologically, *i.e.*, what processes a cross section of the instinctive consciousness would show. Yet the psychology of instinct is at least as important to his general enquiry as the biology of it. The rest of the book is mainly taken up with casuistry. There are chapters on psycho-organic disturbances of sex, on similar disturbances of the instinct of nutrition, on the signification of the so-called "internal hallucinations," on cœnæsthesia and impulse, on cœnæsthesia and delirium, and on the motor power of the organic senses. A general summary of their results would have been serviceable.

It is somewhat late in the day to offer a review of the last work to be noticed—M. Paulhan's "L'activité mentale et les éléments de l'esprit."[1] The book is essentially an attempt to better the associationist psychology, made by an associationist psychologist. The new concept introduced is that of the "system"; we have a law of systematic association and a law of systematic inhibition. The former runs: every psychical fact tends to associate with and to call up psychical facts which are harmonisable with itself; which can co-operate with it for a common end or for harmonic ends; which can form with it a system. The latter: every psychical phenomenon tends to prevent the production, to arrest the development, or to cause the disappearance of psychical phenomena which cannot unite with itself in accordance with the law of systematic association, that is to say, for a common end.

[1] Paris, 1889.

The volume was received with some eagerness on its publication. One hoped to find in it a radical, methodological betterment of the associationist position. As a matter of fact, however, the author does not go very far behind the tenets of the orthodox school. None the less, his book is distinctly a book to be read.

It is, perhaps, hardly necessary to state that the works noticed here constitute only a fraction of the total psychophysical output of the years covered. Several important treatises, as, *e.g.*, M. Fouillée's "Psychologie des idées-forces," have not been mentioned.

E. B. Titchener.

Reviews and Abstracts.

Traité pratique des Maladies du Système nerveux. Par J. Grasset et G. Ranzier, Professeurs à l'Université de Montpellier. Montpellier: Camille Coulet. Paris: G. Masson. 1894. Price 90 fr. Fourth edition, with 122 figs. and 33 plates. Two vols., 8vo, 900 and 1,090 pages.

It is exceedingly difficult to appreciate at its proper value such a monumental work as the treatise of Grasset and Ranzier in its most recent form. The only plan of arriving at even an approximate estimate of its value is to take here and there a section, and regarding such a section as a separate work on the subject with which it deals, to see to what extent it meets the demands which one is accustomed to make on a monograph. For many of the sections viewed in this manner no praise is too high, while others are a little disappointing in their sketchiness.

The plan followed in such a work is of no little importance, and it may be useful if we first indicate what this is, for in this way its defects and its advantages will be obvious. There are in all six parts, the first dealing with brain disease and including circulatory troubles and local diagnosis, encephalitis, tumours and hydrocephalus. The second part deals with spinal cord diseases —myelitis, diffuse myelitis, diffuse cerebro-spinal disease and circulatory troubles; the third part discusses bulbar diseases; the fourth, diseases of the meninges—cerebral and spinal; the fifth treats of nerve diseases and neuroses; and the sixth of nervous manifestations of general diseases. No doubt such an arrangement has much to recommend it. It is probably as comprehensive as any, and to say that it is unscientific in its arrangement is to apply to it a criticism which is equally applicable to every systematic work on diseases of the nervous system that has yet been produced. And the retort generally made to such a criticism is no doubt true, viz., that the state of our knowledge of nervous disease, especially in its patho-

logical aspects, is not yet such as to permit of such a scientific work being produced. The arrangement used possibly leads occasionally to the separation of intimates, as when Jacksonian epilepsy and so-called idiopathic epilepsy are found in different volumes, and, like misfortune, it occasionally makes strange bed fellows, as when tetanus and paralysis agitans are found on neighbouring pages. But in spite of such drawbacks the arrangement is a clear and convenient one, and, as we have said, it is certainly comprehensive.

We have thought it best in order to indicate the general character of the book to take one chapter, and by a brief *resumé* of its contents to show how complete and how well arranged it is. Taking as a good example the chapter on syringomyelia, we find first of all a short but succinct history of this condition from the first use of the word, in 1837, down to the time of publication of the work under notice, and in this is pointed out the curious anomaly that it was known as a pathological condition long before symptoms were recognised as associated with it, and that the clinical recognition of the disease has led to a reconsideration of at least some points in the physiology of the spinal cord. The pathological anatomy of the condition is next dealt with, the appearance of the cord, the number of cavities, their contents and their walls are described and illustrated with a series of beautiful plates, and the relation of the cavities to the central canal is fully discussed, as well as the manner in which they arise. The condition as a clinical one is next considered, and a brief history of its recent discovery is given, and its symptoms both intrinsic, *i.e.*, peculiar to this condition, and extrinsic, those, so to speak, accidentally associated with it, are fully set out. The former comprise the peculiar sensory dissociation, the muscular atrophy and the trophic changes; the latter such conditions as frequently exist in other diseased conditions such as lateral sclerosis and posterior sclerosis. Differential diagnosis is next considered: (1) From such diseases as usually show no sensory impairment, *e.g.*, progressive muscular atrophy, myopathies, amyotrophic lateral sclerosis, cervical pachymeningitis, disseminated sclerosis, tabes, chronic myelitis and compression of the cord by tumour or bone disease; and (2) from diseases in which sensory symptoms are predominant, *e.g.*, neuritis, leprosy and hysteria. In this part is introduced a subsection on the maladie de Morvan, in which are set out the different views held as to the relation of this disease to syringomyelia and leprosy, and while no definite side is taken, it is, we venture to think, indicated that

the authors regard the maladie de Morvan as merely a form of syringomyelia. The article is concluded with a short account of the etiology, the prognosis and treatment. *Ex uno disce omnes*, and if all are not equally good not one can be said to be bad. Further criticism than this is unnecessary except that we may say that the style is characterized by that dramatic clearness and force which brings the type of disease before the reader in all its vivid reality—a characteristic which is so happily evident in the great bulk of French medical writing. The illustrations are good and copious, in no case unnecessary, and as a rule happily chosen to illustrate and complete the text, and the work as a whole is one in which the authors may have a just pride, and in which the reader will have unlimited enjoyment.

JAMES TAYLOR.

Les Altérations de la Personnalité. Par ALFRED BINET, Bibliothèque Scientifique Internationale. Paris: Félix Alcan, 1892.

THE author points out that of late years a large body of observers have been working at pathological psychology through hysteria and hypnotic suggestion. As researches have multiplied numerous discussions have taken place, not only as to the interpretation of facts, but as to the facts themselves. The author's object is not to bring forward his own observations, but to select out of the crowd of researches those which have received confirmation at all hands, can be gathered into one synthesis, and which lead to one conclusion. The time is ripe for such an attempt, for it is a curious fact that a number of observers in various countries, working by diverse means with different ends in view, have consciously or unconsciously arrived at the same conclusion, namely, that underlying the mental phenomena of hysteria and suggestion is a peculiar alteration of consciousness, a doubling or rather a parcelling out of the ego.

In working out his thesis M. Binet takes first, under the divisions of spontaneous and induced somnambulism, cases of dual consciousness, alternating in time.

Spontaneous somnambulism is especially important as its phenomena are not influenced by preconceived notions on the part of the observer. In this condition the subject leads another psychological existence or second life, of which he has no memory on returning to the normal state, and in which he lives

the common life, being open to all ideas and perceptions, and has no delusions. Two mental lives are thus lived side by side; in the first state there is no memory of the second and *vice versa*. Several celebrated cases are referred to. In Félida X., there was loss of memory of the second state when she was in her normal condition, but in the second state she had memory of both the normal and abnormal lives. A change of character also characterises this second condition, which may further be attended by sensory or motor defects. An observation of M. Proust makes known the important fact that the memory during induced somnambulism extends to events of the second state (spontaneous somnambulism). So far these cases have been observed in hysterical patients only, but if changes in memory, which are always more easy to appreciate than those of character, be taken as the touchstone, the author thinks similar phenomena may be observed outside hysteria.

In the next group of cases the second state is altogether different from the normal one; the subject is under the influence of one idea or group of ideas, and is unconscious of his surroundings unless they fit in more or less with the preconceived idea which dominates his thoughts and actions. Mental activity is thus developed in one direction only. This activity has been considered to be unconscious, purely reflex or *machinal*, without trace of conscious thought, judgment or imagination. The author does not accept this view and brings forward data to show the presence of consciousness. Subjects in this second stage may not be able to speak or comprehend speech, but in a patient observed by Charcot this was not the case, and by means of speech the nature of the patient's delusion could be ascertained. The difference from induced somnambulism consisted in the spontaneity of the delusions which could not be guided at the will of the operator. In this class of cases there is dual consciousness, but the second state is attended with delusions. It is, therefore, more difficult in cases of this class to be certain whether, as in those of the first class, memory during the second state extends to all events of preceding phases of this state, but the occurrence of continuity of action during successive phases seems to prove that it does.

Induced somnambulism or the hypnotic state constitutes from the psychological stand-point a new existence—it is the emergence of a new personality. In this form of somnambulism alterations of character are not observed, but the memory shows profound change; first, the subject during the normal life can recall none of

the events of the hypnotic state, and secondly, whilst in the latter he remembers not only the events of former periods of somnambulism but also those of his normal life. Memory in the induced somnambulistic state is thus more extensive and also more exact as regards detail than in the normal life. In this respect spontaneous and induced somnambulism correspond.

The advantage of induced somnambulism is that it enables the experimenter to vary the conditions, and study somnambulistic phenomena more completely. It has thus been shown that the normal life and the somnambulistic state alternate with each other, and that during the former there is no memory of the events of the latter. Gurney has shown that though the subject in the normal is ignorant of the events of the somnambulistic state, and cannot recall them by any effort of the will, yet by putting a pencil in his hand, which is concealed from him by a screen, the hand will automatically write words heard during somnambulism, and further will correctly finish an addition sum begun in the somnambulistic state. The hand writes; the subject is unaware of what is written. All the cases hitherto considered have this in common, that a set of psychological phenomena well co-ordinated amongst themselves form a group outside and apart from the normal consciousness. In natural somnambulism the new or secondary consciousness alternates with or replaces the normal consciousness; in the last experiment it co-exists with it.

Part ii. deals with double consciousness, and is entitled "Co-existent Personalities." Double consciousness is met with under two conditions: (1) in hysterical anæsthesia; (2) in strong concentration of attention on one object. Activities may take place without the ordinary consciousness being aware of them, in the first in the anæsthetic regions and in their correlated nervous centres, and in the second in those parts of the body and their related nerve centres away from which the attention is directed. These secondary activities may show psychical characteristics (intelligence) and so come to form a secondary consciousness existing by the side of the normal one. It is true that automatic actions occur frequently in ordinary life, but they are simply habitual actions, arise from frequent repetition of movements, rarely attain any marked degree of development, and are not therefore well adapted for the present study. Hysterical anæsthesia is to be regarded as a psychical anæsthesia arising from mental dissociation. This can be shown by experiment. In all dealings with hysterical patients due precautions must be observed in order to avoid making suggestions. Hypnotism is unnecessary. All that

is required is to place the anæsthetic limb, *e.g.*, the hand, behind a screen or behind the patient's back and to be careful not to speak to her. Then the hand will carry on single movements imparted to it, will assume the writing position if a pen be placed in it, and further, if it be passively made to carry out a writing movement, on letting it go the word or character may be written. If the hand has been caused to trace a word wrongly spelt, it may, strange to say, when it reproduces the word, be seen to hesitate at the wrong letter and either reproduce the mistake or correct it. Of all these movements, passive and active, the subject is entirely unaware.

In some patients more complicated actions can be evoked than in others. Women, especially those who have been frequently hypnotised, are better subjects than men; probably this is due to the difficulty of eliminating some form of suggestion. The above facts indicate that in hysterical anæsthesia a real perception is gained by the patient, which does not, however, rise into the ordinary consciousness, and since phenomena of memory and judgment have been shown to occur, the existence is presumed of an intelligence different from the ordinary one and acting without knowledge or aid of the latter.

In another form of experiment if the hysterical subject be placed before some test types, but just beyond his distance of distinct vision, although he cannot read them, his hand may automatically write them correctly. The author attributes this to feebleness of the sensory (receptive) organs, and remarks that the "threshold of consciousness" is in hysteria a relative term, excitations below a certain minimum of consciousness being registered unconsciously, and resulting in subconscious actions.

The effect of attention is to exalt the sensibility of the senses engaged in the special work, to lower that of all others coincidently. The distraction resulting from attention is a temporary anæsthesia; hysterical anæsthesia is a permanent distraction. In normal persons, however close the attention, actual anæsthesia is never produced. In the hysterical it is readily produced, and according to Janet this results from "the restriction of the field of consciousness." The secondary consciousness or personality produced in the above ways: (1) perceives sensations over anæsthetic areas; (2) remembers phenomena of similar former states; (3) seems to have a distinct personality; (4) responds extremely readily to suggestion. The identity of this secondary consciousness with that of somnambulism is shown by the state of the memory. So far the most important conclusion reached is the

division or dissociation of consciousness, *i.e.*, the juxtaposition of several psychological states which are never confounded together.

Hysterical patients can often use an anæsthetic limb, and contrary to what one would expect can write when their eyes are closed, and their right hand is completely anæsthetic. As under these circumstances they have no knowledge of what is passing, they judge of how much has been written mainly by lapse of time; thus, if asked to write one character a dozen times in succession they rarely succeed in writing it the correct number of times. The author explains these actions by (1) the preservation of a good visual memory, and (2) the survival of the afferent impulses and motor images in a separate consciousness; he does not think that they can be explained by the theory of a sense of effort conditioned by the outgoing current. Some hysterical persons are unable to carry out any action without the aid of sight, attributed partly to feebleness of visual memory and poor development of secondary consciousness, partly to the fact that light appears to be a necessary physiological stimulus to the enfeebled nervous system. Though the primary and secondary consciousness are separated, yet experiments show that there are important connections between them, that they inter-act one on the other; for instance, an hysterical person asked to recall a name or date may be unable to do so, although it is on the tip of his tongue; in this case it may at once be written without his knowledge by his anæsthetic hand, thus showing collaboration of the primary with the secondary consciousness, and that memory is more extensive in the latter. There is thus no abrupt demarcation, or suspension of all relations between the primary and secondary personalities; each group of psychological ideas influences each neighbouring group, but is only itself cognizant of what is passing in its own domain. Although it has been so far assumed in order to make the argument simpler that the subject is entirely unconscious of what passes in the anæsthetic limb, this ideal is rarely attained. Any excitation of an anæsthetic limb can arouse by means of suggestion conscious ideas; M. Binet thinks that this is accomplished through the intermediary of the secondary consciousness which then influences the primary one. Sensations produced in the anæsthetic limb remain subconscious, but he shows by experiments that they may give rise to ideas, images, false perceptions or hallucinations in the normal consciousness, of whose real origin the subject remains entirely ignorant.

The third part of the book deals with alterations of consciousness (of personality) produced by suggestion.

Fictitious personalities may be created by suggestion. Under the influence of the suggested personality the subject ceases to perceive the external world, and falls under an hallucination. Everything in harmony with the suggestion is evoked, and everything that contradicts it is inhibited, and passes out of consciousness, hence there is amnesia as regards the former life, so that the memories which represent the past existence and form the basis of the ego are temporarily effaced. A division of consciousness is thus made, and a new personality is formed. Two classes of patients exist: in the first their real personality is not lost sight of, but they cannot resist the ascendancy of the operator, the others are completely victims of the illusion suggested. Again, former stages in the life of the subject may be recalled, he may be thrown back into his condition at a past period of his life, and reproduce his behaviour at that age. From the psychological point of view these experiments show that ancient memories which we believe lost because we cannot recall them at will, live on in us; in other words, what we know of ourselves is only a part of what we are. This recall of phases of the past life by suggestion cannot be explained by the law of association of ideas, for if it could be there would be no need of suggestion to provoke the recall. The author argues that if, then, the law of association is not sufficient for this recall, it fails to fully explain the development of the mental life. Causes more profound of whose nature we are hardly aware act to gather up our ideas, perceptions and memories into autonomous and independent syntheses.

Passing on to the discussion of the interesting subject of systematised anæsthesia by suggestion, by which term is meant the condition which results when a suggestion is made to the subject that he shall not perceive a certain object, the author compares this condition to that of spontaneous hysterical anæsthesia, the perception inhibited by suggestion having the same fate as sensations coming from an anæsthetic area, and like them being relegated to a secondary consciousness. Certain facts show that the object inhibited by suggestion is really seen. (1) A square of red paper not seen (obediently to suggestion) calls up an after sensation of the complementary colour. (2) The subject is told not to see one out of a number of similar cards; if they are too much alike mistakes are frequently made, but no mistake occurs if "*invisible*" be written across the card which is not to be seen. Perception and judgment must therefore be present, but are unconsciously exercised, and systematised anæsthesia is preceded by a certain number of subconscious psychological processes.

All negative suggestions do not, however, act by means of a dual personality in this way, although they do so in persons who have been repeatedly hypnotised; in those hypnotised for the first time nothing of the sort occurs—a certain perception is excluded from the sphere of consciousness and that is all—and it must be conceded that much obscurity still surrounds the *modus operandi* of negative suggestion. In discussing spiritualism the author for obvious reasons feels obliged to restrict himself to the facts which consist in the unconscious movements executed by the so-called medium. He brings forward evidence of the existence of dual consciousness in mediums. The peculiar phenomena of spiritualistic manifestations which cause them to differ from those of double consciousness previously studied are due to the fact that the medium is not ignorant of the end in view, like the foregoing hysterical subjects, but is dominated by a powerful preoccupation from his belief in spirits and in the possibility of evoking them. He is thus placed under the most favourable conditions for auto-suggestion. Curiously, however, it is the secondary and not the primary (normal) consciousness which is influenced by the suggestion. Whereas in previous parts of the book division of consciousness is due to hysterical anæsthesia or to distraction, in this case it can be traced to auto-suggestion. The mental modifications are here more complex and less easy to describe than in the first two.

M. Binet concludes: (1) That the elements which normally constitute the ego may be in a state of division or disaggregation.

(2) A consciousness does not cease to accompany these elements, although the primary ego loses knowledge of them.

(3) Sometimes under exceptional conditions, pathological or experimental, these elements are organised into secondary personalities.

The foregoing will give some idea of the main argument and scope of the work. M. Binet, it will be seen, traces the condition of dual or multiple consciousness from the form in which it is most easily observed—alternating states of double consciousness—to those more complex forms in which two or more distinct personalities exist side by side. He shows that this condition may occur spontaneously or be artificially induced. Dual consciousness seems to exist in a more complete and highly developed form when it occurs spontaneously. So far as can be judged due care has been exercised not to admit any observations into the book which are not well authenticated. The whole of the investigation into dual consciousness during recent years, the main facts

of which are given in this book, is exceedingly important from the new light thrown upon the nature of consciousness. Under the conditions mentioned, hysteria and somnambulism, there seems to be a firm basis of fact for the splitting up of consciousness into subordinate parts. To what extent may this subdivision of the mind occur under normal conditions? So far it has been shown to appear in only a rudimentary and imperfect form during concentrated attention in normal persons. One of the most important features of these researches lies in their bearing upon the nature of hysterical anæsthesia, as has been mentioned above.

It only remains to be said that the book gives a most clear and concise account of a difficult subject. The author has marshalled his facts in orderly sequence so that the reader can readily follow and appreciate the line of argument, and the work is one which will amply repay a careful perusal.

J. Michell Clarke.

Recherches Cliniques et Thérapeutiques sur L'Épilepsie, L'Hystérie, L'Idiotie et L'Hydrocéphalie. Par Bourneville, Médecin de Bicêtre, avec la collaboration de MM. Boncourt, Cornet, Lenoir, Jules Noir, et P. Sollier. Vol. xiv., avec 89 figures dans le texte et une planche. Paris, 1894.

The book is divided into two parts. The first gives an account of the work that has been done at the Bicêtre and Fondation Vallée during the year 1893; the second consists of eight essays by Dr. Bourneville and his assistants on subjects of special interest.

Dealing with the first part we find that various improvements have been made in the mode of teaching the children, especially with reference to the education of the eye, the hand, and the faculty of attention. During the year 122 cases were received, 15 died, 33 were discharged, 4 were transferred to other parts of the Bicêtre, and 5 escaped, leaving on December 31, 1893, 512 patients. The causes of death are related, and a table giving the weights of the thymus and thyroid glands is of great interest. The Fondation Vallée is overcrowded, and contains no less than 131 children, but a project is on foot to increase the

size of it by building a pavalion capable of accommodating 100 more patients. In this institution, too, various improvements in the mode of teaching have been made, and exercises for improving the power of the hands and legs have been added.

One of the most important and at the same time interesting essays in the clinical part is that by Dr. Bourneville himself, who contrasts the surgical with the educational treatment of idiots and imbeciles. He analyses eighty-two cases in which craniectomy has been practised, and comes to the conclusion that the results have been slight or doubtful. The operation has been performed by Dr. Lannelongue with the idea of giving free scope to the brain by weakening the resistance of the cranium. He supposes that microcephaly, a generic term under which he describes the different forms of idiocy, is due to premature ossification of the cranial sutures. Dr. Bourneville goes carefully through a series of twenty-two cases, in three of which craniectomy had been practised, and finds no sign of premature synostosis, and he concludes that Dr. Lannelongue's theory is not borne out by pathological anatomy. Moreover, lesions of the brain, which are often found, cannot benefit by the operation. Dr. Bourneville then relates eleven cases in which educational treatment alone has been carried out, and shows very clearly that more good will be effected by this treatment than by craniectomy.

An essay by Bourneville and Cornet on thirty cases of epilepsy, treated by subcutaneous injections of testicular fluid, is also very instructive. Excluding two cases who did not undergo the full treatment, there remain twenty-eight who were injected with the fluid for a period of six weeks to two months. According to M. d'Arsonval, if at the end of six weeks there are no results in the treatment of epilepsy, it is useless to prolong it. Of the twenty-eight patients eight had a slight diminution in the number of their fits, while the remaining twenty had a larger number than before. In no case had there been amelioration of the mental condition. The weight was taken before and after the treatment in each case; unfortunately in several cases the weights have been mislaid, but of ten patients in which these were preserved there was found to be an increase in six, a diminution in three, and no change in one case.

A long essay of nearly two hundred pages on some forms of hydrocephaly, written by Drs. Bourneville and Noir, is interesting. The authors distinguish three forms, viz., simple hydrocephaly, scapho-hydrocephaly, and symptomatic hydrocephaly,

and give the histories of nineteen cases, eleven of which illustrate the first, five the second, and three the third form of the disease. The symptoms generally found are fully described, and the condition mentally, as to language, will, the instincts, and moral and social sentiments, is well delineated. According to the authors, in most cases the effusion of fluid takes place some months after birth, and the child, to all appearance in a normal condition of health, is suddenly taken with meningitis or convulsions. Gradually the head increases in size until the age of 4 or 5 years, when its greatest dimension is reached. Death usually takes place between the age of 3 and 6 years from bronchopneumonia or meningitis. As regards the etiology, all cases of chronic hydrocephalus have as predisposing causes nervous diseases, mental maladies, and alcoholism; the exciting causes being chiefly chronic inflammation of the intra-ventricular meninges or compression of the vessels of the base of the brain by the development of a tumour. The authors do not believe that treatment by trepanning and drainage is of any use, as the result in most cases is fatal; treatment by compression of the head, daily friction with mercurial ointment, and the administration of calomel, aided by the use of salt baths, massage of the limbs, and tonic medicines, being far more successful.

The book is profusely illustrated with wood-cuts of many skulls and brains, and of a few patients who have been under treatment, and shows that Dr. Bourneville and his assistants continue to do much excellent work in their department of the Bicêtre.

FLETCHER BEACH.

Psychiatrie für Aerzte und Studierende. Prof. TH. ZIEHEN. Berlin: F. Wreden, 1894. Pp. ix. 470.

In this work, which forms one of a series of medical text-books, the author treats diseases of the mind on the basis of the psychological doctrines laid down in his "Leitfaden der physiologischen Psychologie," of which an English translation has appeared. One of the marked features of that book was the thorough-going adherence to what the author called the "association psychology of the English," though his system was distinguished from that of English psychologists by the importation of the Herbartian notion of the mutual strengthening or

inhibition of ideas to which the name of "constellation" was given. The first part of the present book, entitled general psychology, extends over 150 pages, and is devoted to an account of the author's psychology with its application to the analysis of abnormal mental symptoms, and it is this part of the book which will be chiefly interesting to neurologists. In it mental symptoms are considered under five heads as disturbances of sensation, of memory images, of intellectual feeling tone, of association of ideas, and of action. Under the first heading illusions and hallucinations are chiefly considered, the latter term being limited to those cases in which there is no actual stimulus external to the body. A novel feature is the division of both illusions and hallucinations into two groups: those in correspondence with the general content of consciousness, and those which have no relation to this content, but arise out of latent or half-forgotten memory images. It is doubtful whether this division, though corresponding to a psychological distinction, is one of practical value.

The section on affections of memory images is chiefly devoted to word blindness and deafness, and these conditions support the author in the opinion expressed in his former book, that the memory images have a physical basis different from that of the original sensations—a view which is gaining ground among psychologists.

In the section on affections of intellectual feeling tone, exaltation, depression, and other emotional conditions are considered. Much importance is attached to irradiation of feeling tone, whereby one element of a mental complex with a pronounced painful or pleasurable tone of feeling transfers this tone to the whole.

The next section, on disturbances of association, is an important one. The relation of increased or lessened rapidity of the flow of ideas to the state of feeling is considered at some length, being of importance in its bearing on the classification adopted later by the author. The subject of incoherence or dissociation of ideas is treated in a specially interesting manner; incoherence is regarded as frequently secondary to other psychopathic conditions, such as increased rapidity of flow of ideas, hallucinations or delusions, violent emotions or weak-mindedness; but the author insists on the existence of a primary dissociation, depending on a disturbance of association paths. Such cases of incoherence are analogous to those cases of aphasia caused by lesions of paths connecting different centres, which may indeed

be regarded as special forms of primary dissociation. This primary dissociation may affect not only the train of ideas, but may occur between the sensation and idea causing defective orientation in regard to surroundings, or it may occur between the ideational and motor elements, giving rise to incoherence of action.

In the section on morbid actions, the author refuses to regard such actions as dependent on disturbance of any special will process, and sees in them only the natural outcome of the disturbed sensory or ideational processes.

In the chapters on diagnosis, ætiology and therapeutics, the author shows his close acquaintance with the more practical side of his subject. The chapter on diagnosis has an excellent outline of symptoms as a guide to the examination of patients.

The chief interest, however, of the latter half of the book will be found in the classification adopted. This differs considerably from those most usually met with in England, and chiefly in the treatment of the simple or primary forms of insanity. Professor Ziehen takes the psychological distinction between cognition and feeling as the basis of his classification. The term "mania" is limited to cases in which the general tone of feeling is altered in the direction of pleasure, a condition the true opposite of melancholia when the alteration is in a painful direction. In both mania and melancholia the change of feeling is primary and the intellectual disturbance present is secondary to the state of feeling.

Cases, on the other hand, in which the primary and dominating disturbance is of the cognitive side of mental life, form a group to which the name paranoia is given, and this includes many, if not the majority of the cases which in England would be labelled "mania." This has four chief sub-groups: acute and chronic simple paranoia, acute and chronic hallucinatory paranoia. In the latter forms illusions and hallucinations are the chief symptoms, delusions developing from these, especially in the chronic variety. In the simple forms delusions predominate, chronic simple paranoia corresponding to the chronic delusional insanity of English writers. These four chief forms have their varieties: acute hallucinatory paranoia, for instance, having varieties depending respectively on the predominance of increased or lessened rapidity of mental processes, of incoherence, of exaltation, or of depression.

The book concludes with an excellent series of plates illustrating the various types of insanity. Professor Ziehen may be

congratulated on this addition to the text-books of insanity. It is valuable especially for the general coherence which has been given to one of the most confused branches of medical knowledge, and this coherence is mainly due to its foundation on the basis of a systematic psychology. Whether this psychology is a true psychology is another matter. Whatever views may be held about it it must be acknowledged that it may form a useful basis for the study of mental pathology.

W. H. R. Rivers.

Hérédo-Ataxie cérébelleuse. Par le Docteur Paul Londe. Paris, 1895. Battaille et Cie. By J. A. Ormerod, M.D.

This is an interesting monograph on a disease which, as yet, is little known. The author begins his work by remarks on "family" diseases, and more particularly family diseases of the nervous system. By a family disease we are to understand one which tends to attack many members of the same stock, and particularly, it would appear, members belonging to the same generation, and which depends not so much on extrinsic causes, whether these be intra-uterine or subsequent to birth, as upon a fault inherent in the germ or introduced into it by procreation. Attempts to classify such diseases according to the rough anatomical divisions of the nervous system (cerebrum, medulla, cord, nerves, &c.) do not give very satisfactory results; hence the author sketches out a classification based on nervous functions and on the "systems" of fibres and centres connected with such functions. Thus the cerebral motor system (highest motor neurons) is affected in the diplegia of Freud and Little, and in the spastic paraplegia of Strümpell; the spinal and peripheral motor system in certain forms of muscular atrophy; the cerebello-spinal system, which is devoted to the function of equilibration, in Friedreich's disease, and in hereditary cerebellar ataxy.

This name (*hérédo-ataxie cérébelleuse*) proposed by Marie, who collected and critically studied cases of the disease, is intended to indicate three main facts:—(1) That it is a family disease; (2) That ataxy, or at least the staggering gait known as cerebellar ataxy, is a leading symptom; (3) That the lesion is in the cerebellum. By this last factor it is distinguished from

Friedreich's disease, where there is also a family ataxia, but where the lesion is spinal. Yet the two diseases are closely related; both consist in a degeneration of the cerebello-spinal system, beginning in Friedreich's disease at the spinal end, in the present disease at the cerebellar. This is illustrated in Menzel's case, wherein both spinal cord and cerebellum were degenerated; the two diseases here co-existed, or rather were exhibited in their proper pathological unity.

The description of the disease is based on the following cases, of which the author gives details in an appendix.

A. Cases collected by Marie; viz., 2 recorded by Fraser (*Glasgow Medical Journal*, 1880), 3 by Nonne (*Arch. für Psychiatrie*, 1891), 8 or more by Sanger Brown (BRAIN, vol. xv., 1892), 3 by Klippel and Durante (*Revue de Médicine*).

B. Original cases; 1 by Brissaud and the author (*Revue Neurologique*, 1894), 2 by the author (*Revue Neurologique*, 1894).

C. Doubtful cases; 2 by Erb (*Neurologisches Centralblatt*, 1890), 2 by Seeligmüller (*Archiv für Psychiatrie*, vol. x.).

There is also the case (just mentioned) by Menzel (*Archiv für Psychiatrie*, 1891); and some extremely doubtful cases by Hervouet (*Gazette Médicale de Toulouse*, 1891) and by Botkin (*Revue Médicale de Moscow*, 1883).

The leading fact in the etiology is the family predisposition. How this predisposition is brought about we know not. Tubercle appears to be not infrequent in such families, and, as in Friedreich's disease, parental alcoholism has been noticed, though whether this is really a cause remains doubtful. Parental syphilis and parental consanguinity do not appear as causes. Little is known as to exciting causes; neither traumatism nor infectious diseases have, in the author's opinion, much influence; in one case (Nonne) the disease followed exposure from shipwreck. The age at onset is comparatively late, usually after twenty; in one case it was past forty. This may explain why the disease appears in successive generations more frequently than does Friedreich's disease, where the earlier onset is more likely to prevent marriage. As to the relative frequency of occurrence in male and female, and of transmission through male or female, we can as yet hardly be certain.

Stated generally, the symptoms are as follows:—An unsteadiness in walking and standing appears gradually and slowly progresses, being sometimes accompanied by pains in the legs and loins; after a variable time (usually one to three years), the movements of the hands become uncertain, and the speech and

the eyes become affected in a manner that will presently be described. The tendon reflexes are preserved or exaggerated; sometimes there is actually contracture of the limbs. The mind sometimes deteriorates. The disease is progressive, though there may be remissions; death is mainly due to intercurrent disorders.

The author proceeds to a more minute description.

Motor Symptoms.—The gait is cerebellar, *i.e.*, the patient walks with feet apart, swerving and swaying from side to side, as if intoxicated. This is mainly due to difficulty in maintaining the equilibrium of the trunk, but there may also be, particularly in advanced cases, a true inco-ordination of movement in the legs. In standing there may also be unsteadiness, but this is not increased by closing the eyes (no "Romberg's symptom"). The earliest defect in movements of the hands is a wavering or hesitation as the hand nears its mark; then a tremor or oscillation which shows itself towards the end of a voluntary movement. Fine actions, particularly that of writing, are the first to suffer distortion; later a tremor affects coarse movements, such as that of lifting a cup. Apart from this tremor of the hands, which may be compared to that of disseminated sclerosis, the patient exhibits involuntary jerky movements, like those of chorea, in his limbs, trunk, head or face. Another peculiar motor symptom is a prolongation of muscular contraction, so that the patient cannot, when he wishes, let go an object he has once grasped. The face exhibits what may be called an exaggeration of associated movement. Thus, during any natural facial movement, but particularly while speaking, the occipital frontalis is thrown into violent action, and the eyes open widely, so that the patient has a staring, astonished look; the naso-labial fold becomes exaggerated, the mouth may even be drawn into a tetanic grin. The speech is remarkable—not syllabic, nor hesitating and elisive: but jerky and explosive, words and syllables being brought out with sudden and unnecessary vehemence. The voice is usually low, guttural and monotonous; or it may break suddenly into high notes. The head is unsteady, particularly when the patient is excited; or there may be a definite spasm of the neck-muscles, causing torticollis. In some cases there has been marked vertigo. The tendon reactions are normal or exaggerated; the muscular sense is normal, the muscular strength is normal.

Sensory disturbances, if present at all, are vague and subjective, *e.g.*, headache, pain in the loins, numbness in the legs, in one case shooting pains.

Trophic disturbances are practically absent; it seems doubtful

whether spinal curvature and club-foot may not sometimes occur. The expression (except for the over-action of the facial muscles just mentioned) is dull and dejected; memory and intelligence are sometimes dulled, the disposition may be irritable.

Remarkable *ocular symptoms* may occur; thus, as regards the fundus oculi, optic atrophy of very various degrees has been observed, and its concomitants, amblyopia, and contraction of the field of vision. As regards ocular movements—during fixation there is a sort of instability of the eyeballs, a condition of irregular, jerky movement, resembling the choreiform movements of the limbs rather than true nystagmus. In several instances there has been a paresis of the R. external rectus, sometimes of the R. superior rectus. Sanger Brown noted a peculiar droop of the upper eyelid, existing only when the face was at rest, and giving place during animation of the face to overaction of the levators and occipito-frontalis, which causes a peculiar stare. There are no particular *visceral troubles;* menstruation comes on late, probably from a general backwardness of development; certain difficulties of deglutition and of micturition recorded by Sanger Brown are explicable (our author thinks) by an ataxia of the muscular movements concerned, in fact, ataxia and instability constitute the keynote of the symptomatology.

A few remarks, which must be regarded as provisional, are made as to the possible stages and varieties of the disease. There may be prodromata of a vague character, "neurasthenic" they may be called; the first stage (ataxia) is marked by the reeling gait; the second (astasia) by such increase of inco-ordination as prevents walking or standing alone, and by the appearance or aggravation of the disease in the hands; the third (impotence) by general helplessness and disability from aggravated ataxia, rigidity, optic atrophy, &c. The author thinks that two principal forms may exist, consisting of ataxia with, and ataxia without, ocular symptoms, and that there may be also characteristics special to each family, such as the peculiar ptosis noticed by Sanger Brown, the facial asymmetry, mental deterioration, &c., by Nonne, vertigo by Fraser, hysteria by himself.

About the pathology much remains to be learnt. Our author holds, with Marie, that the essential anatomical fact is atrophy of the cerebellum. But there are only a few *post-mortems*, those by Fraser, Nonne, Menzel. Nonne found a widespread atrophy of the nervous system; Menzel's case showed degeneration of the cord as well as of the cerebellum. Still (as is pointed out in a chapter on the physiological pathology) the rough resemblance

between the main symptoms of this disease, and those produced by other lesions, experimental or morbid, of the cerebellum, serves in some sort to designate the cerebellum as its seat. So far as we yet know, the character of the lesion is a pure and simple atrophy or disappearance of nerve elements, and therein differs from the cerebellar atrophy which follows traumatism or infectious diseases, wherein is found evidence of old meningitis and a sclerosis of the nervous centres from overgrowth of interstitial connective tissue.

The differential diagnosis from other kinds of family nervous diseases is considered. Obviously the nearest relative is Friedreich's disease. To save space we may, perhaps, refer our readers to a previous number of BRAIN (vol. xv., 270), for the clinical resemblances and differences of the two diseases. Dr. Londe lays special stress on the condition of the knee-jerks—absent in Friedreich's disease, present or exaggerated in hereditary cerebellar atrophy. Whether the knee-jerk is *invariably* absent in Friedreich's disease pure and simple we are not prepared to affirm. But, however that may be, we welcome as most suggestive the theory he advances, that both diseases consist essentially in a family tendency to degeneration of the cerebro-spinal system, which strikes in the one disease mainly at the cerebellum, in the other mainly at the cord, and may sometimes, as in Menzel's case, involve both organs together.

Herter and others on Intestinal Putrefaction in Certain Neuroses.

HERTER[1] refers to his previous researches on intestinal putrefaction in epileptics,[2] and says that further researches have shown that excessive putrefaction in the intestines is met with in a variety of morbid states.

To determine the amount of this putrefaction he now makes use of Baumann's discovery that the various aromatic substances resulting from this putrefaction are after absorption eliminated through the urine as ethereal sulphates, and his observations are based on the quantitative estimation of these products.

In forty cases of epilepsy, two-thirds of the cases of grand mal showed distinct evidence of excessive putrefaction; in a few cases

[1] *New York Medical Journal*, January 27, 1894.
[2] *Ibid.*, September 3, 1892.

the excess seemed to correspond with the number of seizures, and reduction of the sulphates by treatment seemed to diminish the number of attacks.

Five out of eight cases of melancholia showed an increase of sulphates, but the author questions whether the increase might not be due to partial starvation, which is often present in these cases, for it is well known that inanition increases the putrefactive products.

In two cases of typical mania the sulphates were normal.

In six out of eight cases of chronic Bright's disease there was a marked increase of ethereal sulphates, and a considerable excess in many cases of simple anæmia, though in many the amount was normal.

In intestinal indigestion there was often an excess of putrefaction, and in intestinal flatulence, perhaps related to chronic catarrhal inflammation in the duodenum, indigo-blue was frequently met with in the urine.

"Many of the symptoms were related to the disturbances of nutrition that resulted in excessive elimination of uric acid, such excess being frequently found in intestinal indigestion associated with an excess in the ethereal sulphates, or with indigo-blue, or existing by itself."

The author suggests that the symptoms depend, to a large extent, upon the action of substances formed in the intestine during the putrefaction of proteids coincidently with the formation of the aromatic substances.

Excess of ethereal sulphates in the urine is evidence of excessive putrefaction of proteids in the intestine except when there is suppuration without free drainage; or except when certain drugs, as creasote and salol, are being given.

The regular occurrence of more than a trace of indigo-blue in the urine is pathological; it does not vary in proportion to the ethereal sulphates, but is due to a special form of putrefactive decomposition.

Intestinal putrefaction has as its most common symptom intestinal flatulence, but is not related to epigastric pain or tenderness, emptiness, or nausea.

Constipation and starvation increase putrefactive products in the urine, and epilepsy, acute melancholia, and chronic Bright's disease, are usually associated with excessive intestinal putrefaction.

Drug treatment of this putrefaction is unsatisfactory; but a diet of raw beef and milk, and the exclusion of vegetable nitrogen

did good, and such diet reduces the excess of uric acid observed in many cases of intestinal indigestion, and part of the relief might be due to this.

Treatment of the intestinal condition was useful in epilepsy, and begun early in children might prevent the establishment of the epileptic habit.

Among those who spoke in the debate which followed the reading of this paper, Dr. L. C. Gray had got good results with a combination of salol, bicarbonate of sodium and pancreatin, and used also calomel and subgallate of bismuth, and Rochelle salts, in small doses for a long time. He regretted that the author had not given the amount of uric acid he found in these cases, as in his own cases, where patients were put upon a nitrogenous diet, there was always an excess of uric acid, and dietetic treatment had not given good results in his hands.

Dr. C. L. Dana had also found an excess of uric acid in almost all cases, but his treatment had been unsatisfactory. He recommended naphthalin for neurasthenic headaches to the extent of 60 to 80 grs. in a day.

Dr. W. H. Thomson agreed as to the frequent presence of intestinal derangement in epileptics, and their improvement when it was corrected.

He had not found any diet-list of use, but he believed in salol, or bismuth with benzoate of sodium.

Dr. L. Emmett Holt had had experience of intestinal indigestion in children, and for this he cut off carbohydrates and put them on a milk and meat diet, with an occasional dose of calomel when improvement was rapid.

Macpherson,[1] writing on disinfection in some forms of acute insanity, notices the very powerful effects of purgatives in aborting or cutting short attacks of recurrent insanity, and the very frequent association of gastro-intestinal troubles with melancholia, and argues that certain poisonous ptomaines resulting from putrefactive changes in the intestines might get into the circulation and so affect the nervous system.

He accordingly, in such cases, began by washing out the stomach and clearing the bowels with calomel with or without other purgatives, and on the second day of treatment administered naphthalin, which he prefers to naphthol. Nitrogenous food was also as far as possible eliminated from the dietary, and peptonised gruels given in place of custards. He gives details of four cases and a summary of his results in some thirty cases.

[1] *Journal of Mental Science*, January, 1893.

Anæmia was prevented or removed to a very marked extent. Weight generally increased steadily. Pigmentation and dryness of the skin disappeared. Sleep was promoted and became natural and refreshing, and this occurred even when sulphonal had previously failed, and he thinks that the naphthalin suppresses the causes that prevent normal sleep.

Some mild melancholias were cut short, but more severe cases, though not shortened, were greatly improved, as "the mental distress and motor restlessness of melancholia rapidly disappeared, the suicidal cases became quieter, and the tendency to impulse in all the cases was almost entirely removed," and he claims for the treatment that it modifies the prominent troublesome and distressing symptoms of acute melancholia, and enables one to dispense with the use of sulphonal and hypnotics.

SINGER[1] finds that there is a distinct connection in time between excessive intestinal putrefaction and certain skin diseases, such as urticaria, acne vulgaris, and pruritus. That the two sets of troubles frequently appear together and may disappear spontaneously together, while treatment of the intestinal trouble has an undoubtedly beneficial influence on the skin trouble. He agrees with Herter and Macpherson in estimating the amount of intestinal putrefaction present from the quantity of aromatic sulphates passed in the urine, and he thinks that excessive intestinal putrefaction is not infrequently present without other obvious signs of dyspepsia.

While indican is often in excess in the urine in idiopathic urticaria, it is not met with in the erythema due to parasites, &c.

He has derived great advantage from the internal administration of menthol, which acts as an antiseptic, anti-fermentative, tonic and digestive, while purgation alone does no good.

He considers that the products of intestinal putrefaction are absorbed into the blood and come to the skin and act either on its vessels or its nerves.

Whatever the explanation, the skin troubles are connected with the digestive troubles, and give us a means of estimating their extent.

FREUND,[2] whose paper on "Autointoxikations-Erytheme" follows that of Singer, has apparently had very similar experiences. He has also seen the use of calomel produce good effects on the skin, but on the whole he prefers ol. menthæ. pip., which, in his hands has quickly caused indican to disappear from the urine, and rendered the stools free from odour.

[1] *Wiener Klinische Wochenschrift*, Jan. 18, 1894.

[2] *Ibid.*, prev. ref. p. 39.

As to Herter's original researches on intestinal putrefaction in epilepsy, I have already said in BRAIN (spring and summer number, 1893, pp. 252 to 256) that he is in my opinion mistaking an effect for a cause, and that the uric acid, which, by its effects on contraction of arterioles and blood pressure deranges the cerebral circulation and produces the fits, interferes at the same time by contracting the arterioles and hindering the circulation, with gastro-intestinal digestion and allows putrefaction to take its place.

So that the fits and the intestinal putrefaction are two results of an excess of uric acid in the blood, and this accounts for their being seen together, and also for the excess of uric acid in the urine which Herter acknowledges is often found with them.

Exactly the same argument will apply to melancholia and Bright's disease, in both of which there is an excess of uric acid in the blood, causing that most common symptom in these diseases—high blood pressure.

Then, again, I have repeatedly pointed out that obstructed capillary circulation in the skin similarly produced, may account for numerous alterations in the function and nutrition of the skin from mere paleness and dryness, eruptions, &c., up to Raynaud's disease, atrophy and destruction; so that I can easily account for the relation observed by Singer between intestinal putrefaction and certain skin eruptions; here again we have two results of the same cause, and for several years past I have been treating diseases of the skin by clearing the blood of uric acid, and so improving its circulation.

The one thing common to all these conditions, call them neuroses or what you will, is obstructed capillary circulation and high-blood pressure, and, if I am right in my interpretation of the facts, it is common to them all, because it is the cause of all.

The obstructed circulation is in every way proportional to the amount of uric acid in the blood and urine, and is almost certainly due to an excess of this substance in the blood stream; all the other symptoms and phenomena are the result of this, and may consequently occur together, in various combinations.

Thus in migraine there is not only headache but also cold skin and extremities, scanty saliva and urine, fœtid motions, mental obscurity and depression, deficient excretion of water from the lungs, all these being results of obstructed capillary circulation, and this, again, is the result of an excess of uric acid in the blood, as is shown by the fact that all the circulatory troubles and symptoms are proportional to the excess of uric acid that is passing in the urine.

And the migraine and all its concomitant signs in the skin and extremities, glands, intestines, kidneys, brain and lungs, can be completely prevented by diminishing the intake and formation of uric acid, so that there is no excess in the body available for solution in the blood, and this can be done by diet, without the use of any drug whatever.

So that, while there is no doubt that these observers have been able to do good in these cases by attacking the intestinal condition, this is simply due to the fact that every improvement in digestion and absorption of food means a fall in the alkalinity of the blood and a diminution of the uric acid it can hold in solution, just as dyspepsia, however caused, means a rise in the alkalinity of the blood and an increase of the urate it holds in solution, so long as there is a supply of the substance in the body to be dissolved. In a word, the intestinal condition, though a result of excess of uric acid in the blood, tends, when it has come into existence, to keep up that excess, and make matters worse.

But none of these results prove that the epilepsy, headache, or melancholia on the one hand, or the skin troubles on the other, are due to the intestinal putrefaction. The only simple and complete explanation is that all these things are alike the result of one cause—the altered circulation which is common to all the organs of the body.

I can easily understand the good results obtained by Macpherson, for the calomel cleared the blood of uric acid, and the improving digestion tended to keep it clear, while the lowly nitrogenous diet introduced little or no fresh uric acid into the body. My researches on the causation of anæmia also completely explain both its presence and its rapid cure in his cases.

Herter's results with meat and milk are more difficult to understand, as meat introduces additional uric acid; and this had, apparently, been noticed by others, who failed to obtain good results with meat; but, as I have also pointed out, a dose of uric acid may cure a uric acid headache, because it raises the acidity for a time, and throws uric acid out of solution in the blood. Herter himself specially notices the plus excretion of uric acid in the urine that is frequently found with excessive intestinal putrefaction, but he has not had my experiences of the effects of uric acid on the circulation, and so he draws what I believe to be an erroneous conclusion from these researches, as well as from his original investigations on epilepsy, which were undertaken to test the truth of my statements as to the excretion of uric acid in that disease.

For further facts and cases bearing on these points I must refer those who may be interested to the second edition of my work on "Uric Acid in the Causation of Disease,"[1] where I think they will find an explanation of the observed facts, accounting for the time relation between these disturbances, as well as for their connection with very numerous disease processes having a similar origin.

Since the above was written, Drs. Herter and Smith have published a further paper on excessive intestinal putrefaction,[2] and I am interested to see that in this they have much more to say about uric acid, and indeed acknowledge that it may cause several of the symptoms they are speaking of, quite apart from any excess of intestinal putrefaction, so that they are led to recommend the treatment of uric acid; and, though I cannot agree with their mode of treating it, I am glad to see that they are coming to acknowledge its power. I will merely point out, in conclusion, that the calomel, which several of these authors find so useful, does not act as a direct intestinal antiseptic; but, forming an insoluble compound with uric acid, it clears the blood of that substance, with the result that a free circulation with natural secretion and healthy digestion and absorption throughout the whole intestinal tract is again possible; and not the intestines only are affected, but every tissue throughout the whole body participates in the improved circulation and its results, so that you get diuresis diaphoresis, increase of saliva and of water given off from the lungs, as well as an improved cerebral circulation and consequent removal of the "neuroses"; and similar results follow the taking of calomel, whether given for excess of intestinal putrefaction or in more normal conditions.

A. Haig.

Theodor Kaes on the Medullated Nerve-Fibres of the Cerebral Cortex. (*Archiv fur Psychiatrie*, xxv. Bd., 3 Heft, s. 695.)

This contribution concerns the comparative degrees of abundance of medullated nerve-fibres in different parts and layers of the human cerebral grey cortex, and in some measure also deals with these as influenced by age.

[1] This is now out of print, but a third edition is in course of preparation.

[2] *New York Medical Journal*, June, 1895.

The results given in this paper are limited to those drawn from minute examination of the brains of two men, of a somewhat inferior class of society, dying of acute phthisis—one at the age of 18 and the other at that of 38.

Although brain-weights at different ages show that the growth of the brain, as indicated by weight, has nearly attained its completion by the age of 20, and that the weight remains about stationary from then until the age of 50, after which it declines, yet the development of the mental faculties and psychic activities does not occur in a parallel manner, since they stand at the height of their power from the age of 40 to 60. Instances of aged persons of particularly comprehensive and effective mental power show that the brain is still capable of active development in even the later years of life. But in microscopic measurement of separate parts of the brain and cortical layers, and in systematic critical estimation of the various cortical districts as concerns the abundance of medullated nerve-fibres in the several layers, we have a more subtle and more precise expedient than in simple weighing.

The examination of the two brains (aged 18 and 38 at death) showed that the formation and development of the cortical fibre-system advances much from the age of 18 to 38; and, moreover, that the formation and increase of fibres, and therewith the further augmentation of the sum total of the mental powers and of their effectiveness, is not yet exhausted at the latter age.

Between the ages of 18 and 38 the brain-convexity gyri increase considerably at the summit, this increase concerning chiefly the association-system of the second and third layers. On the other hand there is no increase (possibly even a lessening) of the lateral breadth of cortex at the brain-convexity. A general increase also occurs at the median and inferior surfaces of the brain, and, as a rule, equally distributed in the several layers, although there are some curious contrasts and contrary relative conditions on the two surfaces on comparing the first layer with the second and third.

Many interesting comparisons are given of fine cortical measurements, but these are too detailed for reproduction here.

As to the question: Which are the brain-districts richest in nbres?—while Vulpius believes the right anterior central gyrus to be so (in which he found the largest number of thick tangential fibres), and while Tuczek maintained that position for the central gyri, so rich in thick fibres, and for the occipital lobes with very numerous differently-thick fibres, Kaes indicates in a general

way the motor cortical field (central gyri and back-end of frontal gyri), the temporal gyri (especially their posterior part), and the occipital, as being the districts which, at the age of 38, are the most advanced according to the criterion of fibre-value; whereas, at the age of 18, the temporal lobe is far behind the other two great regions in development of nerve-fibres.

The paracentral region possesses the greatest abundance of large fibres; the occipital outstrips all other cortical regions in the number of fibres and the early development.

In the motor zone, Kaes believes that he has proved that the posterior paracentral region in its general development is almost as plenteously endowed with fibres at the age of 18 as at 38.

The district poorest in fibres is the cerebral convexity at the frontal tip, and nearly like it in this respect is the gyrus rectus.

W. Julius Mickle.

Sur les connexions du ruban de Reil avec la corticalité cérébrale.—Dr. and Madame Dejerine. Société de Biologie, April 6, 1895.

After describing the various views as to the distribution of the fibres of the fillet (*ruban de Reil*), the authors refer to three cases of its ascending degeneration, in which the degeneration could be traced as far as the anterior corpora quadrigemina and the lower part of the optic thalamus, but no further. They found the posterior segment of the internal capsule intact, as well as the ansa lenticularis, the body of Luys, the globus pallidus, and the commissure of Meynert. These results confirm the experimental researches of Vejas, Singer and Münzer, and of Mott. Dr. and Mme. Dejerine also find descending changes in the fillet in old-standing lesions, involving the lower and posterior part of the optic thalamus and the fibres lying behind and external to the body of Luys; but in all these cases there was simply a slow atrophy, not a degeneration in the proper sense of the term, although it may lead ultimately to the complete disappearance of the fibres. This atrophy diminishes from above downwards, from the thalamus towards the nucleus of the columns of Goll and of Burdach, and is proportioned to the degree of destruction

of the fillet and to the time that has elapsed since the original lesion.

In a case of a lesion in the sub-thalamic region which had lasted for twenty years, the atrophy, well-marked in the tegmentum of the cerebral peduncle and in the upper part of the pons, was much less marked at the lower border of the pons, while in the medulla, the inter-olivary stratum and the internal arcuate fibres of the opposite side were only slightly affected. The authors believe that the atrophy spreads slowly from the periphery of the neuron towards its cells of origin, and is analogous to that produced in the central segment of a bundle of fibres in the brain or medulla which has been divided by a lesion. In other words, they follow Monakow's view that the fillet is mainly composed of fibres which arise from cells in the nuclei of the columns of Burdach and Goll. They do not think it probable that any cells of origin are situated in the thalamus. When the lesion is in the pons, the descending atrophy is much more marked than when the lesion is in any situation above the pons. This depends not only on the fact that the destruction of the fillet is often more complete in these cases, but also that the seat of the lesion is nearer the cells of origin. They further agree with Monakow and Mahaim, in opposition to Flechsig and Hösel, in the view that the fillet is only indirectly connected with the cortex of the cerebrum. In nineteen cases of degeneration of the cerebral cortex of the Rolandic region and the parietal lobe, they found no case of degeneration in the fillet. At the most there was a slight diminution of this system due to a slight diminution in the size and not in the numbers of the fibres. They have also examined three cases of very old lesions which, besides affecting the cortex, had also destroyed the insula, the operculum, the putamen, the caudate nucleus and the globus pallidus, leaving, however, the internal capsule and the optic thalamus absolutely intact. In these cases there was absolutely no effect on the fillet. They conclude, therefore, that there is no direct connection between the nuclei of Goll and Burdach and the cerebral cortex, and that the sensory bulbo-cortical tract comprises two neurons, an inferior or bulbo-thalamic neuron, represented by the median fillet, and a superior or cerebral neuron, connecting the thalamus with the cerebral cortex.

ALEXANDER BRUCE.

Putnam on the Relation of Infectious Processes to Diseases of the Nervous System. Pathology and Etiology. (*Am. Jour. Med. Science*, 1895, p. 254.)

(1) The author inquires into the pathogenesis of the nervous affections which are obviously of infectious origin; (2) investigates the claims of the affections which are only suspected to be of infectious origin; (3) investigates the contributive causes which make certain parts of the nervous system peculiarly liable to suffer from infectious influences; and remarks that the direct effects of infection are not to be feared more than the prostrating influence of the original struggle.

Out of 5,000 cases of affections of the nervous system he found that about 500 had had, within a year before the onset of the illness, some infectious disease. About 80 per cent. of these affections were post-influenzal and post-syphilitic (even excluding the cases of paretic dementia and ataxic tabes with a syphilitic history, which numbered respectively 29 and 71); the rest, in order of frequency, were cases preceded by diphtheria (53), typhoid (47), scarlatina (14), malaria (14), measles (9), gonorrhœa (8).

In order to consider, first of all, to what extent the results could be explained without referring them to the direct action of the infective agent, an intimate acquaintance with many influences, of which the following are the chief, is required:

(1) The nervous system has certain natural proclivities to certain forms of disease in the production of which the infectious process only joins hands with developmental or inherited tendencies, or accentuates the habitual strains of life [laws of "devolution" (Jackson); laws of "degeneration"; disproportion between waste and repair (Edinger)]. Our knowledge of these tendencies is still far from complete.

It is not only the typical psychoses and neuroses which can arise in this way, but also some of the acute and chronic degenerative affections, such as symmetrical polyneuritis and the different forms of tabes. These factors seem, however, to be called out more strongly by certain infectious processes than by others, and made to operate in more or less definite ways, so that, clinically, the connection between the infection and the final disease, though indirect, may be close.

(2) The infectious processes may give rise to secondary disorders of nutrition, of cardiac, vascular, digestive, sclerotic and metabolic origin, and a primary infection is liable to become

complicated at any part of its course by a secondary infection with its own train of consequences. Poisonous substances are being formed in, and constantly eliminated from, the body; when this elimination is checked or arrested, as often happens in the course of infectious processes, an auto-intoxication takes place which bears its share in the production of symptoms [Bouchard (16)]. Such researches as these should make us cautious as to how to explain the clinical and anatomical effects of infection. Bouchard comes to the conclusion that there are two classes of influences at work in cholera infection: first, the poison primarily produced by the specific organism; second, a secondary poisoning due to uræmia (itself a complex and variable cause), and that each of them have their own symptoms, which may succeed or alternate with each other.

The principal effects produced by the infective agents on the nervous system are:

A. A series of *primary disorders* is induced, not directly due to gross lesions, but to the toxic action of one or more of the *soluble elements* of the (often complex) poison.

In this class of primary toxic affections belong the symptom-complexes of tetanus, rabies and whooping-cough, a portion of the cerebral, spinal and meningeal symptoms of acute infectious diseases, and perhaps many of the characteristic symptoms of diphtheria, Landry's paralysis and chorea. Symptoms of peripheral nerve disorders *may* be due to poisoning without recognisable lesions. Ether is well known to be a nerve poison, and if injected into the tissues in the neighbourhood of a nerve, degeneration will follow. On the other hand, when an exposed frog's nerve is kept moistened at one point with ether, its power of conduction may be inhibited for at least forty minutes at a time (Bowditch), and yet on the removal of the ether the nerve may resume its functions as if uninjured.

The signs characteristic of the nervous affections following infectious processes are due far more to the toxic substances which are developed in the course of bacterial action than to the micro-organisms themselves.[1] The formation of tubercle and abscess, and the exudation of purulent meningitis imply the presence of micro-organisms, and bacteria are found now and then in the brain, spinal cord and nerves, under various circumstances, without its being possible to say as yet what their relation is to the lesions accompanying them.

[1] See *Therapeutic Gazette*, 1894, p. 185; also Flexner (Toxalbumins, &c.), *Medical News*, August 4, 1894.

It is only in the case of lepra that the bacteria may be said actually to invade the nerve-cell or nerve-fibre (Babes [9] and others), and even here the occurrence is not constant (Kuhne, Zambaco-Pacha). Ordinarily, the bacteria lie in the lymph spaces. The presence of histological cell-changes after infection does not necessarily imply the direct action of bacteria, for they may occur in a typical form where only sterilised and filtered cultures have been used, as has been shown by Welch (93) for the case of diphtheria. (See also Flexner, *loc cit.*)

B. The *peripheral nerves* suffer from disorders of nutrition due to—

(1) Infiltration with toxic substances; (2) Interstitial and vascular disorders; (3) Diseases of their trophic centres. As compared with neuritis due to mineral poisons, the infectious neuritis is perhaps more frequently due to the second of these causes (Cramer), but the distinction is not absolute.

C. The *cerebral* and to a less extent the *spinal membranes* become breeding-places for colonies of bacteria. Possibly meningitis is occasionally due to the irritation of soluble toxines, as in some cases of scarlet fever [Bendel] (*Wiener med. Presse*, 1894, No. 23).

D. Focal (generally hæmorrhagic) non-suppurative *lesions of the brain, cord,* or *nerves* may be set up by either bacteria or their toxic products. We know as yet but little of this matter except through experiments with animals, though acute lesions of this sort are met with which are supposed to be of infectious origin. Bacteria of many kinds have indeed been found in the central nervous system, though rarely in man, as the obvious cause of focal inflammation, with the important exception of abscess and tubercle. Characteristic bacteria are also claimed by Pfuhl to have been found in the foci of encephalitis following influenza. We now know that specific toxines are capable of exciting focal lesions, and we are looking out for clinical evidence indicating a direct causal connection between the soluble poisons of influenza, diphtheria, variola, scarlatina, typhoid, gonorrhœa (Leyden, Hayden and Parmentier, Dufour), and the focal myelitis which sometimes follows them.

Enriquez and Hallion (*Soc. de Biologie*, April 14, 1894) have also found that the toxines of diphtheria injected under the skin of dogs may cause, *if the dose is not too large*, intense congestion of the grey matter of the cord, with hæmorrhages, and foci of inflammation characterised by formative irritation of the neuroglia and destruction of nerve elements. Symptoms suggesting a

similar process were also seen in an ape. Animals are, perhaps, more subject than man to *toxine myelitis*. At any rate, localised destructions of the grey matter of the cord have been found by Gilbert and Leon (*Semaine Méd.*, 1892, p. 65), and more recently by Thoinot and Masselin (*Rev. de Méd.*, June, 1894), after inoculation with virulent cultures of the bacillus coli communis; by Bourges (*Soc. de Biologie*, 1893) after injections of erysipelas cultures (streptococcus pyogenes ?); and by Vincent (*Arch. de Méd. expérimentale*, 1893), after inoculation with typhoid bacillus. Roger, also (*Ann. de l'Institut Pasteur*, 1892), has produced a poliomyelitis by inoculation of rabbits with a streptococcus obtained from erysipelas, and his observations are particularly interesting from the fact that this result only attended a certain phase of virulence of the organism, which he was not able to reproduce at will.

It is probable that acute myelitis in man is sometimes of infectious origin. The possibility of secondary infection should, of course, never be lost sight of. Auché (*Semaine Méd.*, Nov., 1894, *Soc. de Biologie*) found a secondary streptococcus infection to be the probable cause of a myelitis following variola.

E. Diffuse lesions of nutrition of nerve-cells and tracts are being found more widely in proportion as our means of investigation improve, as witness the changes in the posterior columns observed by Bikeles in diphtheria; many of these may, however, be due to œdema or disorders of circulation (see Edinger, *Sammlung klinischer Vorträge*, No. 106).

The earliest lesions in cases where none were supposed to be found are described by Golgi (41) as occurring in *rabies*. The nerve cell shows impairment of its nutrition by peculiar changes affecting the size and contents of the nucleus. The cell-protoplasm, the endothelium of the vessels, and the neuroglia take part in the change, which is one first of irritation, then of reaction and repair. He denotes the whole process an "encephalo-myelitis parenchymatosa." The irritative stage is associated with active hyperæmia. He asserts that the histological peculiarities are not pathognomonic. They only mark where the poison has struck, and perhaps indicate the severity of the blow. It is especially noteworthy that most of these changes, as observed in rabbits, were found before the stage of complete paralysis had been reached, and similar lesions were in fact noted in the cerebellar cortex, within four or five days after inoculation and before any symptoms had appeared. One is reminded by this statement of the peculiar form of parenchymatous neuritis observed by Gombault in guinea-

pigs, as a result of chronic lead-poisoning, at a period when the animals appeared to be perfectly healthy. Golgi further notes that these changes are very similar to those which he had discovered in chorea.

Minute changes such as Golgi describes are only to be accepted as pathological when made by trained observers and under the most favourable conditions of preparation. Even then it might not always be easy to say that we were not dealing with the effects of fatigue or of œdema, and still more difficult to distinguish between the action of specific toxines and that of the toxic products of disordered nutrition. A good deal has been made of the toxic origin of vacuolation of the nerve cell [see Campbell (20)]. Vacuolation has, however, been noted under such varying conditions that one must wait to see whether it indicates anything more than one of the ways in which nerve cells regularly degenerate. It certainly affords no means of distinguishing between bacterial poisons and others (Afanassijew,[1] Campbell).

The distribution of the lesions which are noted by Golgi is more important. There would have been no special reason to suspect that the brain and cerebellum as well as the medulla and spinal cord would be involved in rabies, and the fact that this is found to be so, assuming that the observation is correct, is an additional reason for thinking that many poisons which seem to be very limited in their action, are really universal nerve poisons, and will excite widespread lesions if time enough is given them.

When we pass beyond these minute parenchymatous lesions, the change which seems most characteristic is vascular congestion with infiltration of leucocytes, often associated with hæmorrhage, and perhaps due to endothelial changes, at least in part. This tendency has been often discussed elsewhere; but the œdema so produced may in itself be a cause of some of the associated nerve-lesions. It might cause degeneration of nerve-fibres, by pressure or disturbance of circulation, and such destruction of nerve cells as is seen in poliomyelitis. When the hæmorrhage and exudation are of infectious or toxic origin there is probably infiltration of poison into the nerve elements and the other tissues in the immediate neighbourhood of the affected vessels; but whether this infiltration precedes the congestion, or is a necessary factor in its production and localisation, is unknown. (See Flexner, *Med. News*, Aug. 4, 1894, on similar affections of other organs.)

[1] "Acuter u. chron. Alcoholismus," *Beitrage zür Path. Anat. u. Allgem. Pathol.*, 1890, p. 443.

The next question is with regard to the influences which increase the liability of the nervous system to suffer from the infectious processes—the contributive causes of disease.

The question is divisible into two parts. The first concerns the influences which lessen the power of resistance of the body as a whole to invasion by the organisms of disease, the second concerns only the influences which increase the vulnerability of the nervous system to the poisons produced by the organisms.

The distinction is an important one. A healthy person, with intact mucous membranes and vigorous nervous centres (see Bouchard, Chorrin, Gamaleia, Buchner, Gley), might resist the onset of infection, while even a healthy nervous system might not be able to withstand the action of a specific virus. Indeed it is open to question whether the poisons do not sometimes affect healthy more strongly than enfeebled nervous centres, for the response to the action of a poison (*e.g.*, that of tetanus) is often in reality a sort of reaction which may imply a normal vigour. A difference is also likely to exist between the character and degree of the functional response to acute intoxication, and the liability on the part of the nervous system to suffer local lesions of nutrition, which experiment has shown to attend toxine poisoning as well as the direct action of bacteria.

Strong nervous centres are probably as liable as weak ones to exhibit the *toxic symptoms* of acute infectious poisoning, while feeble ones are more prone to show *disorders of nutrition* under the added strain of the presence of the poison, especially if this is reinforced by impaired circulation or œdema.

Among the post-infection disorders of the nervous system there is none so common as *neuritis*, usually, though not always, symmetrical. The infectious toxines are capable of causing neuritis, but we probably include under this name a number of other affections. Such are: (1) the neuritis attending the first stage of an infectious process before there has been time for the development of general disorders of nutrition; (2) the post-infectious neuritis, in the production of which general disorders of nutrition and a variety of other causes play an important part; (3) the acute, usually fatal and hæmorrhagic, neuritis of unknown origin. Nor should it be forgotten that what we have considered as neuritis is often associated with nuclear and diffuse spinal lesions. Paralysis of the peripheral type may be unattended by demonstrable lesions, and this fact has lent support to the view that the spreading forms of paralysis (Landry) may belong to the same category.

According to Ross[1], Leyden[2] and others, there is no marked difference in the clinical history of some cases of spreading neuritis and Landry's disease, and the mere absence of demonstrable lesions does not justify us in setting apart the latter cases as belonging to a special group. But other pathologists (Hun[3]) hold that so long as there does appear to be a class of cases where the disease is generally fatal, and yet no definite lesions are found, there is nothing gained by lumping them, for the sake of pathological unity, with demonstrable cases of neuritis. Putnam thinks that the mere absence of demonstrable lesions in a given case of poisoning cannot be taken as proof that the pathogenesis of the symptoms is not the same as in another case where lesions were found. That which is characteristic is often the primary action of the poison rather than the effects of the lesions, even when lesions are present. The history of tetanus and diphtheria, and perhaps rabies, gives warrant for this assertion, for in some instances of these diseases few or no lesions are discoverable, while in others the lesions are more extensive than the symptoms would have suggested. Goldscheider (*Berl. klin. Wochenschr.*, June 4, 1894, p. 546) takes the position that the presence or absence of demonstrable lesions in Landry's paralysis is a matter of slight importance. Jolly (*ibid.*) takes the position assumed by Hun.

Part of the symptoms of acute hæmorrhagic (infectious?) neuritis are unquestionably of peripheral origin—those, namely, which are due to the pressure of the endoneural exudation. It is, however, far from certain whether death, in these cases, is due to the neuritis or to a poisoning of the nerve centres.

Leyden and others have noticed that in some of these cases changes in the spinal cord are discoverable (swelling of axis-cylinders). The author described such a case where death occurred at the end of a week from respiratory paralysis (*New York Journal of Nervous and Mental Diseases*, 1887). Edinger (*loc. cit.*) thinks that this swelling of axis-cylinders is often referable to œdema, but it is probable that it at least implies an impairment of resistive power of morbid origin.

The more chronic forms of neuritis which follow known infection are probably toxic in origin; but it is uncertain whether the poison is identical with that which caused the primary infection, or is due to secondary disorders of metabolism; the

[1] "A Treatise on Peripheral Neuritis," 1893.

[2] *Zeitschr. für klin. Med.*, vol. xxiv.

[3] *New York Medical Journal*, May 30, 1891.

stimulants given during the illness may be a contributive cause (Starr).

The peripheral nerves (or their trophic centres) are singularly sensitive to poisons of many kinds. Ross enumerates forty causes of neuritis, and among those recently noted are puerperal conditions [Moebius (62), Bassette (11), see also *Nouvelles Arch. d'Obstet. et de Gynécol.*, 1894, No. 9,] erysipelas [Grasset, Lew,] septicæmia [Lloyd and Riesman (58)], pyelonephritis [Dana], suppuration of the pleura [Fiessenger (35)], gonorrhœa [Engel-Reimers, *Jahrbücher der Hamburger Staatskranken Anstalten*, 1890].

The lesion is not precisely the same in all these cases. After acute infectious diseases the morbid process may centre around the blood-vessels, and is said to be rather interstitial than parenchymatous [Cramer (22)]. The spinal cord is apt to be involved with the nerves in these cases. In some instances the parenchymatous degeneration may be due to the pressure of œdema. In the acute (hæmorrhagic) form, which is supposed to be of primary infectious origin, the trunks of the nerves and the nerve-roots may be involved, as well as the peripheral portion. Different varieties of degenerative neuritis are also described, such as the "periaxillary" and "segmental" forms (see Cramer). Research may show that where an irritant organic poison is circulating in the blood, capable of causing neuritis, the endothelium of the vessels will show changes like those described by Golgi as occurring in rabies.

Cerebro-spinal membranes.—The symptoms are probably due partly to the exudation itself, partly to the irritation of the sensitive membrane, partly to the absorption of toxic products. The organism of both the epidemic cerebro-spinal meningitis—the sporadic forms not associated with suppuration elsewhere—and the meningitis which sometimes accompanies pneumonia, is the so-called pneumococcus, or micrococcus lanceolatus. The history of this important bacillus has already a voluminous literature, but interesting analyses are given in the papers by Netter (63), Welch (93), Foà (*Zeitschr. für Hygiène*, 1888), and in the research by Flexner and Barker (37) into the nature and causes of a recent epidemic of cerebro-spinal meningitis. This (pneumococcus) form of meningitis seems to be by far the most common variety even of those occurring independently of epidemics of pneumonia. Out of thirty such cases Netter found sixteen to be due to this organism, while the largest number induced by any other single organism was six. Out of forty-five cases of primary meningitis reported by other observers

and collected by Netter, thirty were instances of pneumococcus infection.

Flexner and Barker think it more probable that the micrococcus passes into the digestive tract and thence, by way of the blood, to the brain; the injection of cultures does not excite meningitis in animals unless made directly into the cerebral cavity.

The high importance of inflammations of the antrum, nasal sinuses, and ethmoid cells for the introduction of organisms to the cerebral membranes, has of late been commented on by many writers—Beck, Macewen, Dmochowski (29), Neumann and Schaeffer (64), Allport (4). (See Macewen's "Pyogenic Diseases of the Brain" for the mode of invasion of the organisms, the formation of abscess, and the origin and spreading of thrombosis and meningitis.) Meningitis may also be due to the bacillus of typhoid. (Tictine, Adenot, Drews.)

It is possible that the poison of scarlet fever and measles may occasionally be the direct cause of meningitis (see Bendel, *loc. cit.*); *acute, primary, hæmorrhagic encephalitis* occasionally follow various forms of the acute infectious disorders, and also occur without known infection, though perhaps not independently of a toxic cause, a possible source of error in the diagnosis and even in the pathological examination; the author reported a case of post-influenzal disease (75) which was believed to be an example of this disease, but subsequent examination showed that nothing more than œdema could be made out with certainty. An additional case is reported by Koenigsdorf (*Berlin. klin. Wochens.*, 1892, No. 9).

Relation of the *chronic spinal scleroses to infectious processes.*—We know how frequently locomotor ataxia and lateral and mixed scleroses follow syphilis. The relation of disseminated sclerosis to several infectious processes, and especially to malaria, is almost equally evident. We do not know what is the nature of this connection, but it clears the way to note again the natural proclivity of the nervous system to become so diseased. Thus we may admit other causes besides the specific virus. The case of syphilis in its relation to tabes, &c., is a case in point. It contradicts all that we know of the action of syphilis, and the usual seat of its lesions, to assume that it is the direct cause of these degenerative scleroses.

Gummatous inflammation may indeed give rise to many of the symptoms of tabes, and this may be cured; and it is true, also, that various modes of treatment may help ataxics for

a time. But the relation of syphilis to locomotor ataxia is to be explained only on the assumption that some new (toxic ?) influence is brought into play, perhaps on a predisposed nervous system.

Sclerosis of the posterior columns occurs as a sign of structural weakness in Friedreich's disease;[1] it follows ergot, pellagra, and lathyrus poisoning; it occurs as a part of the degenerative process in chronic paralytic dementia; as a consequence of morphine-poisoning in dogs [Alt(5)]; and its outbreak is precipitated by such processes as influenza and trauma.[2]

Posterior sclerosis is sometimes associated with progressive pernicious anæmia, and perhaps with other affections which originate in auto-intoxication (from the digestive tract ?).

The first communication on this subject was by Lichtheim, in 1887 (*Sixth Congress für innere Med. zu Wiesbaden; Verhandlungen*, p. 84); but, in 1893, Dr. W. Minnich published a careful monograph on the subject, and gave full reports of all the cases observed up to that time. The author had previously observed a series of cases, one or two of which were certainly cases of pernicious anæmia, and in 1891 he published a paper under the title "A Group of Cases of Sclerosis of the Spinal Cord associated with Diffuse Collateral Degeneration, occurring in Enfeebled Persons past Middle Life, and especially in Women." (*New York Journal of Nervous and Mental Diseases*, February, 1891.) Various partial causes were noted, and among them lead-poisoning and chronic diarrhœa.

It is true that Minnich declines to admit that his cases are of the same order as tabes, or ergotism (Tuczec; Grünfeld, *Arch. für Psych. u. Nervenkrankheiten*, vol. xxi., p. 618); but he is unable to explain them on a different basis except that they seemed to originate in foci of disease related to the vessels, and he admits them to be probably toxic in character.

His interesting paper also contains an account of another form of change in the posterior columns which seems to occur in

[1] What is true of posterior sclerosis is true, also, of the other spinal scleroses and degenerations and the mixed forms, among which latter Friedreich's disease belongs. (See a paper and discussion by Strümpell and others. *Weiner klin. Woch.*, 1894, No. 4, p. 69. Mendel here remarks that even multiple sclerosis may rest on a hereditary basis.)

[2] Cf. Morton Prince, *Neurol. Assoc.*, 1894, and Klemperer, *Zeitschr. für klin. Med.* xvii., 1890. The interesting observations and ingenious theory by Obersteiner and Redlich (95) does not close the discussion as to the pathogenesis of tabes. The argument that the posterior roots are squeezed at their entrance into the cord by a thickening of the membranes, of syphilitic origin, fails, as the authors recognise, to account for many other associated lesions of tabes, and to recognise the rapid increase in symptoms which attends general depressive influence.

some cases of pernicious anæmia, probably toward the end of life, and to be due to œdema. The rest of the cord is affected to some extent [*cf. Bikeles* (95)].

Lichtheim refers to other conditions besides those here mentioned, which contribute to disease of these tracts, and suggests the effect of sensory excitations.

Edinger's theory is that repair does not keep pace with waste. (*Sammlung klinische Vorträge*, No. 106.)

It is true that just as there are differences between the different forms of neuritis, so, also, the posterior sclerosis which follows syphilis is probably not identical with that due to the other causes mentioned. The ergot-sclerosis, for example, is not so malignantly progressive, and is, perhaps, anatomically different; and there are other peculiarities of the different varieties, some of which are pointed out by Raymond (78) and by Friedheim (38). But there is a strong bond of resemblance connecting them all. There is even some evidence, apart from the important case of Friedreich's disease, that tabes may be a member of the so-called neuropathic family (especially when it develops in the later years of life).

Among the less common lesions of syphilis the author mentions a form of myelitis closely resembling the ordinary transverse dorsal myelitis. The disease is of slow development, and the spastic element is strongly marked, in proportion to the paralysis and the loss of sensibility, which are relatively slight. The patients may improve, especially under the influence of an energetic specific treatment, while, at the same time neither the symptoms nor the improvement point to typical gummatous disease. This form was first described by Erb, in the *Neurologische Centralblatt*, No. 6, 1892, under the heading of "Syphilitic Spinal Paralysis." (See also Kowalewsky, *Neurologisches Centralblatt*, 1893, No. 12, and Kuh, *Deutsche Zeitschrift für Nervenheilkunde*, 1893, iii. 6.)

Another form of syphilitic disease is that where more or less typical lesions of syphilis are associated with those of tabes: important, because cases of this sort are perhaps those where specific treatment has some results. Dinkler (*Deutsche Zeitschrift für Nervenheilkunde*, 1893, iii. 4 and 5) reports a striking case of this kind, and gives references to the earlier literature. (See also Sachs, *New York Med. Journ.*, January 6, 1894.)

Diphtheria.—Conflicting views respecting the nerve lesions are held by Gowers, Arnheim, Hochhaus, and Cramer. Gowers and Arnheim describe the neuritis as parenchymatous, while

Hochhaus and Cramer think that in diphtheria (as in the other infectious processes) the primary change is vascular and interstitial, and Hochhaus refers many of the symptoms to pressure of œdema. Gowers quotes an interesting observation by Boissarie (*Gaz. Hebd.*, 1881, Nos. 20 and 21), describing a series of cases of paralysis of the palate, eyes, limbs, heart, "perfectly like that which occurs after diphtheria, and accompanied by albuminuria. The remarkable fact is that in these cases of primary palsy there was no history of preceding sore-throat, and in several of the cases distinct diphtheria followed the paralysis, which lessened during the throat affection. Some of these cases of primary palsy seem to arise by infection." At the same time there occurred a series of cases of severe diphtheria. We may now assume that careful examination of the throat would have shown that the bacteria were really present, even where no soreness of the throat had occurred.

The experimental study of the nerve lesions of *diphtheria* (see Gowers, 2nd ed.) has given conflicting results. Good observers have repeatedly failed to discover histological changes to account for paralyses experimentally induced, and although changes have been found in man, especially in the peripheral nerves, yet they have been occasionally missed (even by recent observers; see Bikele's paper in *Obersteiner's Arbeiten*, Heft 2, 1894), and perhaps represent only a terminal stage in the process.

Quite recently an experimental research by Stscherbak (86) has given more positive results. The changes which he observed can be divided into (1) those involving the peripheral nerves, consisting in well-marked degeneration, which on the whole corresponded in position and intensity to the paralytic symptoms, and (2) those involving the spinal cord, membranes, nerve-roots, and the muscles. The former he regards as constant; the latter rather as incidental than typical.

The diseases of the nervous system which are suspected, but not proved, to be of infectious origin, are, especially, beri-beri, poliomyelitis, Landry's disease, certain forms of myositis, neuritis, myelitis, some of the cerebral palsies of children, chorea, disseminated sclerosis and other cerebro-spinal and spinal scleroses, amputation neuritis, herpes zoster.

The grounds on which a disease may be suspected to be of infectious origin are, when bacteria are found in the tissues; when the outbreaks occur in epidemics or are related to seasons or locality; when vascular and histological changes characteristic of the infection are found; or specific toxicity of the blood and

urine; or if the disease has become much less frequent since the introduction of antiseptics.

On this latter ground the intractable *neuritis following amputation* has been suspected to be of infectious origin [Page (70)]. It is true that even when great care is taken to prevent infection, amputation neuritis still occasionally occurs, but it should be remembered that at the best surgical asepsis can be but relatively perfect.

As regards the lesions to which the nervous system is liable as the result of infectious disease, there are some which are more or less characteristic in view of its structure and liabilities, and others which it shares with other organs and tissues of the body. Of the latter class are the vascular lesions; hæmorrhage and exudation, which are of vaso-motor origin or due to changes in the vascular walls such as are liable to occur in any part of the body. Where well-marked evidences of these vascular lesions occur it is common to consider them not only as evidence of infection, but also as the cause, rather than the consequence, of the parenchymatous lesions with which they are associated. Goldscheider (40) supports the theory of the infectious origin of acute hæmorrhagic neuritis or myeloneuritis and encephalitis, as well as of *poliomyelitis*. In the latter disease the signs of congestion and leucocyte infiltration are prominent, and a case published by Putnam in the *Journal of Nervous and Mental Diseases*, 1883, not included in Goldscheider's (40) or Siemerling's (85) lists, agrees with this view. The author expressly notes that the wasting of the ganglion cells was not by groups, but "depended on their position with regard to the centre of inflammation," and that "the process is not one which affects ganglion cells materially more than other tissues in their neighbourhood." He suggests "that the original morbid change was one affecting the vessels and the circulation."

Another argument in favour of the toxic theory of poliomyelitis is that this disease is certainly related to the acute (often hæmorrhagic) polyneuritis, and that for this disease we find still more evidence of toxic origin. In both affections we see the strong vascular element in the onset; in both the occasional relation to acute infectious disease, and with neuritis the relation to beri-beri.

As regards the analogy between poliomyelitis and neuritis, several instances of the combined occurrence of the two diseases have been published [Gowers (43)]. In Putnam's case of central myelitis the clinical evidences of severe and widespread peripheral neuritis were unequivocal.

"Poliomyelitis" may include several affections which ought to be separated; or, in other words, that several different poisons are liable to affect the anterior horns of the spinal cord, exciting results which, though in the main alike, may differ in detail.

The experimental evidence is in favour of this view, as well as the varying characters of the so-called epidemics of poliomyelitis. (See especially Caverly, *Medical Record*, Dec. 1, 1894, and Macphail, *Medical News*, Dec. 8 and 22, 1894). The number of cases studied was 126, and the symptoms of some of them were so peculiar that, although the diagnosis of "poliomyelitis" was considered as the most probable one, it was not regarded as perfectly satisfactory. [See also Siemerling (85)].

As regards the infectious origin of *Landry's paralysis*, all we can say is that it occasionally follows recognised infectious diseases, and that its abrupt onset and rapid course, often at least unattended by gross lesions, point definitely to some toxic cause. There is no reason at present for calling this cause bacterial, any more than poliomyelitis.

The theory of the infectious nature of *chorea* is interesting, but still far from being proved. The subject is well presented in papers by Pianese (73); Dana (24); Berkley (*Johns Hopkins Reports*, 1891), and others.

Pianese's investigation dates back to 1891. The only point which needs to be mentioned here is that he found in the cervical portion of the spinal cord, the medulla oblongata, and the cerebellum—but not, it would seem, in the brain proper—a bacillus which he considers as specific, and which he was able to cultivate and use for experimentation. Injections of pure cultures into animals gave rise to motor symptoms and often death, but apparently did not cause anything that could be called chorea. In the animals experimented with, the bacillus was found again in various parts of the nervous system, including the brain. Pianese has also examined the blood in a number of cases of ordinary chorea, and in one of them succeeded in finding this bacillus.

Dana's (24) first article was published in BRAIN, in 1890, and contained a report of accurate investigations into the pathological anatomy of chorea. His examination confirmed and defined those of previous observers (Hanford, BRAIN, 1889-90, p. 129). It seems clear that the most obvious change consists in vascular congestion, dilatation, sometimes leading to thrombosis or capillary hæmorrhage, and that widespread degeneration of the nerve elements may also occur, besides subacute meningitis. In Dana's (26) second paper, besides changes of these sorts (though in this case the signs

of vascular congestion were less marked), a small number of micro-organisms resembling the diplococcus lanceolatus were found in the deeper layers of the pia mater and the superficial part of the cortex. The case was not one of typical chorea, however, and the number of micro-organisms was to few too warrant their being considered other than incidental.

Berkley describes a series of minute changes, chiefly in the membranes and vessels. Areas of focal inflammation were found in various parts of the brain, medulla, and pons, and among other conditions which are important as indicating the infectious character of the disease, there was a peculiar endarteritis, which, as Berkley says, is rare in persons of twenty-seven, except from an infectious cause. Micro-organisms were not found, but deposits of detritus were observed, which resembled the remains of bacteria. In the second part of his paper, which describes the case of a choreic dog, Berkley compares, in adjacent columns, the changes which he found with those given by Babes (8) as characteristic of diphtheria, in order to show the analogy between the two processes.

If ordinary chorea turns out to be of infectious origin we must suppose that the bacillus which causes it is present in an inactive state in large numbers of persons, for the disease may develop instantly, in consequence of fright, and some sort of predisposition must be present, for the disease is one which occurs chiefly at certain seasons of the year. We cannot say whether the lesions point to a toxic agent due to bacterial action or to a toxic agent induced by some form of disordered metabolism, or due to retained excretions.

With regard to the other affections, the argument in favour of the infectious origin is mainly that they are apt to follow other general diseases admitted to be infectious, like the acute exanthemata, but the exact significance of that argument is uncertain. One or two recent observations suggesting a relation between malaria and *disseminated sclerosis* have been published (Marie, Torti and Angelini), and Putnam has seen a striking example of the sort. A man in middle life and of good health was attacked suddenly with characteristic and progressive intention-tremor and scanning speech immediately after a sharp malarial attack. Under treatment by arsenic there was a slight improvement only.

The argument with regard to *herpes zoster* consists essentially in its tendency to occur in epidemics—though it must be admitted that this tendency is by no means strongly marked—and

in a somewhat marked liability to follow other acute infectious diseases. [See Weigert (92), Schäffer (81), Kaposi (51), Wasielewski (90)].

The infectious theory of *epilepsy* calls for critical examination. The argument that the attacks are temporarily arrested by some intercurrent infectious malady, which might be supposed to furnish antitoxines, to the blood, cannot be seriously advanced by anyone familiar with the history and therapeutics of epilepsy [see Féré (33), Marie (60), Quériand (76), Séglas (84), Lannois (55)].

The origin of *beri-beri* is still a matter of doubt; some recent observations (Scheube, Glogner; *Virch. Arch.*, vol. cxxxii. p. 50), suggest that it is a protozoal disease, analogous to malaria. Glogner calls attention to the periodicity of the outbreaks of the symptoms, and claims to have found changes in the blood and an organism resembling the plasmodium. (Some recent observers attribute the disease primarily to imperfect hygiene, like scorbutus.)

Another means by which it is thought that the infectious character of these nervous diseases may be tested is the chemical and physiological study of the blood and urine. [Albu (2), Séglas, Fisichella, Ewald, Bouchard, D'Abundo (27), Régis and Chevalier-Lavanol (79), Voison and Peron (89). See also *Boston Medical and Surgical Journal*, September 14, 1893, and *Archives de Neurologie*, 1893, p. 240.] It is evident that before much can be done here, the toxicology of normal urine must be thoroughly understood, and for this reason the recent publications of Bouchard are of decided value, though the conclusion which he reaches will doubtless require modification. Bouchard maintains that if men are not poisoned by the products of their own tissue-metamorphosis, it is because the poisons which are constantly being formed are as constantly carried off from the blood in the urine. He finds seven toxic substances in normal urine, each of which has properties of its own. The first is diuretic in its action, and consists practically of urea. This is only toxic in a considerable dose, and is in general prophylactic in its tendency, since by increasing the quantity of urine it helps to carry off the other substances. The second ingredient is narcotic in character, and its chemical nature is not known, except that it is soluble in alcohol. The third is also soluble in alcohol, and has the peculiarity of causing salivation. The other substances are characterised by their power of causing convulsions, contraction of the pupil, reduction of heat. One of the convulsive agents is potash. In uræmia, &c., all these poisons are increased in quantity.

Summary.

It is important to separate (*a*) the symptoms and lesions which we can assert to be directly due to the specific toxines of the various infectious diseases, from (*b*) the effects of anæmia, œdema, intoxication, from disordered metabolism, on the one hand, and on the other hand from (*c*) the affections which spring into existence because the nervous system was prone to them, either from peculiarities of structure, anatomical surroundings, circulation, and the like; or because other causes had been at work, to which the infective poison only contributed new effectiveness for evil.

In the first class (*a*) we can fairly place the symptom-groups of tetanus and rabies; and with reasonable probability the chill, pains, paræsthesias, acute delirium, disorders of micturition, and other symptoms which characterise the outbreak of many acute febrile disorders, though it must be admitted that we know little or nothing positive about their pathogenesis.

The focal hæmorrhagic encephalitis and myelitis occasionally observed after influenza and other infectious processes is probably due to the action of the specific poison on the tissues and the vessels. Acute poliomyelitis and acute hæmorrhagic neuritis may be of the same origin, though these opinions need further substantiation.

As regards tetanus and rabies, it is not probable that the anatomical lesions which have been found after death (Golgi and others) are actually the anatomical substratum of the characteristic symptoms. They may have been simply of correlative origin.

Post-infection meningitis is generally of purulent character, and implies the presence of micro-organisms; but the non-purulent form bears a relation to infectious disease which is in need of further study.

The paralyses of diphtheria are probably attributable to the direct action of the poison, but, as in case of tetanus and rabies, it is by no means certain that the lesions sometimes found (though often missed) are the true cause of the symptoms. It is more probable that the first and characteristic effect of the poison is to excite a chemical or molecular change, and that this alone is strictly characteristic. Sometimes the paralysis passes away too rapidly to be attributed to neuritis. The lesions, when present, may be many and various, and the neuritis, which is the commonest form, is of varying types. (Meyer, *Virchow's Archiv.*, 1881, vol. lxxxv. p. 181.)

The lesions met with in meningitis, and in a certain sense those characteristic of tuberculosis and syphilis, are directly due to bacterial action.

In the following cases the influence of cause *a* is more or less combined with those of causes *b* and *c*.

The question as to the origin of the post-infectious multiple neuritis is one of great interest. The most obvious position to take is in favour of its direct dependence on the infective poison; but, in view of the general fact of the intolerance of the peripheral nerves to poisons of all sorts, it is fair to suspect that the post-infectious neuritis may sometimes be due to toxic substances developed from impaired metabolism and sometimes to anæmia and œdema (Class *b*).

It appears to be occasionally possible to induce neuritis by experimental inoculation with diphtheritic cultures, and the form and sequence under which the diphtheritic paralyses clinically occur suggest the action of a specific poison. If, however, the forms under which neuritis typically occurs are considered, it becomes obvious that natural proclivities of the nervous system are at least contributive causes.

The variety of conditions under which the spinal scleroses—both disseminated and system scleroses—occur, suggests predisposing tendencies of one or another kind, common to all and acting in addition to the various special causes. The recent observations of Obersteiner (95) and Redlich, that the narrowing of the posterior nerve-roots at their point of penetration through the pia mater suggests a mode by which slight pressure (as from syphilitic meningitis) could set up the ascending degeneration characteristic of tabes, do not afford a complete explanation of that disease. This theory leaves unnoticed the close analogy between the syphilitic tabes of the lateral columns and that of the posterior columns. The occurrence of the peripheral neuritis met with in connection with syphilitic tabes, but rarely seen at other stages of syphilis, points to the presence of some poison as yet undiscovered, and strengthens the probability that the posterior column degeneration of anæmia and ergot-poisoning, as well as that of syphilis, is mainly of toxic origin.

The complex nature of the infective poisons is shown by the varying relation between the toxic symptoms of the first stage (fever, delirium, pain, &c.) and those of the later stages.

In influenza the fever may be of trifling amount and duration, and yet the post-infectious diseases severe and lasting; in cerebro-spinal meningitis the fever is often slight. Again, it has been

found possible, experimentally, to neutralise to a certain extent the fever-producing element in bacterial poisons by special anti-toxines. (V. Centanni and Bruschettini, *Deutsch. med. Wochenschr.*, 1894, No. 12, p. 270.)

Finally, it is probable that a routine examination, by the best modern methods, of the central and peripheral nervous system, after infection, would show changes to be present far oftener than is now supposed. The studies of Golgi in rabies lend support to this view.

As an additional contributive cause of lesions, fatigue may be mentioned, since the experiments of Hodge (50) show that this is capable of exciting manifest changes in the nerve cells. It is a question for further investigation whether or not fatigue acts more forcibly on nerve centres poisoned by the products of infectious processes.

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(92) Weigert. "Herpes Zoster. Realencycl. II. Aufl. Art Entzundan."

(93) Welch. "The Micrococcus Lanceolatus," *Bull. of Johns Hopkins Hospital*, 1892, xii.

(94) Zambaco-Pacha. "Etat des nos Connaissances actuelles zur la Lèpre," *Semaine Méd.*, 1893, No. 37.

(95) "Arbeiten aus dem Institut für das Studium des Nervensystems, &c." Wien, Heft 2, 1894.

RECENT ARTICLES ON PARAMNESIA.

(i.) **Sur les Paramnesies.**—At Lalande, *Revue Philosophique*, November, 1893.

(ii.) **La reconnaissance des phenomènes nouveaux.**—Bourelon. *Ib.*, December, 1893.

(iii.) **Observations sur la fausse mémoire.**—Dugas. *Ib.*, January, 1894.

(iv.) **A propos de la paramnesie.**—J. le Lorain. *Ib.*, February, 1894.

(v.) **Note sur le paramnesie.**—Lapie. *Ib.*, March, 1894.

(vi.) **L'impression de "l'entièrement nouveau" et celle du "déjà vu."**—Duejas. *Ib.*, July, 1824.

(vii.)—**La paramnesie ou fausee mémoire.**—J. van Biereliet. *Ib.*, July, 1894.

(viii.)—**Sulla paramnesia, o falsa memoria.**—T. Vignoli. (Cited by Soury.) *Ib. Ib.*

Little attention had been paid to the subject of paramnesia since the studies of Kräpelin and Burnham until the last year, during which a series of articles have appeared in the *Revue Philosophique* and elsewhere, which, if they afford no definite

solution of the problem, have at least recalled attention to its salient features.

To recapitulate, Kräpelin[1] adopted Sully's[2] classification of illusions and hallucinations of memory, parallel to illusions and hallucinations of sense-perception, confining himself to the study of the latter, *i.e.*, total falsification of reminiscence. This he subdivided into (*a*) simple or direct; (*b*) associative; (*c*) identificatory delusions of memory. Paramnesia, in its modern sense, is confined to the third class only. The essentials of true paramnesia are, absolute and total recognition of the scene or situation ("ce n'est pas une autre perception, c'est identifiquement la même"), a painful emotional state varying from mere *ennui* at always living through familiar sensations to acute terror and suffering, and, in some cases, illusory prevision of the future. It is essentially a phenomenon of normal life. Pathological cases of paramnesia are only extreme forms of what occurs under normal conditions, and "the further study of paramnesia will probably furnish admirable illustrations of the way normal mental activity passes over into abnormal." (Burnham, *Am. Jour. of Psychol.* ii., 1889.) It occurs in the waking state, in perfect sanity, full possession of the critical faculities, and yet makes good its distinctive character. Strictly speaking about 30 per cent. are subject to it. It is unaffected by sex and education, is most frequent among the young, or in persons with a lively imagination, is evoked by fatigue or by unusual excitement, and we may note that the phenomenon occurs, *e.g.*, in crowds, or at theatres, under mass-influence.[3]

The theories advanced in explanation of these facts do not impress one as conclusive. The earliest that we have in paramnesia, a real memory of a former existence, may be rejected. Nor does Maudsley and Wiegan's double functioning of the cerebral hemispheres carry conviction. Anjel had a pretty theory of delay between sensation and perception, the mind is affected by sensations before it co-ordinates them; but this would not necessarily produce paramnesia. Lalande (i.) falls back on the double representation of the same image—a momentary interruption and resumption of perception, seeming in the rapidity of thought to be prolonged or projected indefinitely, and further appeals to the "telepathie" sense in support of his opinion. We

[1] "Ueber Erinnerungs Fälschungen, Kräpelin." *Archiv. für Psychiatrie.*

[2] "Illusions." J. Sully (International Scientific Series).

[3] Cf. Farde. "Psychologie des Sectes et des Foules." (*Revue des Deux Mondes*, 1894.)

cannot deny, he says, that there exists an abnormal and unconscious perception, a telepathic sense, due either to the intensification of known senses, or to the rudiment of another, as far beyond them as sight is beyond touch. The unfamiliar excitation of this sense makes us perceive twice over. It can similarly be aroused by dreams, passion, the hypnotic state, and other still unknown physiological agents. Whatever be the surmise adopted, the key to paramnesia lies in the existence of a double perception, at first unconscious, and subsequently conscious.

To this Dugas (iii.) replies that paramnesia is only complicated by the introduction of telepathy, and suggests that it is a special case of double personality and perceived at the moment of occurrence. He finds a cause in spontaneous auto-hypnosis, and, unlike Lalande, believes paramnesia to be a neurosis.

Le Lorain (iv.) follows with a short critical note which endeavours to reduce the phenomena to the category of associative memory, but he fails to account for cases of "true" paramnesia.

Meantime Vignoli (viii.) was applying these several theories to cases of paramnesia under his immediate observation. He adduces several striking personal experiences, and brings them under the laws of normal psychical processes, telepathy and hypnotism being superfluous. He proposes three causes: (i.) the awakening of mental images, ideas, and feelings by association; (ii.) the rapidity of thought; (iii.) the constructive power inherent in mind and imagination, and insists strongly upon the latter.

In his second note (vi.) Dugas remarks that the sensations which gave rise to paramnesia are usually characterised by banality, producing an undefined feeling, a sense of familiarity, which Fouillée (introduction to *La Venèse de l'idée de temps*, Guyau) had compared to colour sensations. May there not be two classes—*sensitifs* and *intellectuels*—the former having a vague sentiment of time in memory, the latter its localised representation? It is worth noting, adds Dugas, that paramnesia seems to be allied to coloured hearing, and the faculty of self-abstraction from time and space, as though connoting a general "sensitivity" of temperament.

The last two notes (ii. and vii.) concur in showing the influence of attention on paramnesia. Bourdon proposes to examine the clear state of mind which accompanies cognition, instead of the more or less obscure conditions of recognition. He finds that in any series, the perception of known objects is keener than of un-

known, *i.e.*, it is usually the former which are perceived vividly. Hence, when the unknown, or "new" sensation surges up with unusual vigour, we assume that it is known; we tend to "recognise" what provokes attention and interests us. Hence a close relation between attention, perception (or cognition), and recognition, productive of confusion.

Van Biereliet also assumes that the ideas and images which suggest the illusion of paramnesia must be unusually vivid—the subjects being in an absorbed state in which the usual landmarks afforded by the memory of motor images are in abeyance. In this state of "passive" attention new images may arise as freely as represented images—the field of consciousness is momentarily narrowed. The illusion of familiarity persists until the new "sencept" is complete when, normally, its elements are seen to form part of a *new* representation. Motor images are specially predominant in children, and where they are temporarily in abeyance the psychical mechanism must inevitably be disturbed, and illusions follow.

* * *

[Note Flete.]—It must be remarked in conclusion that the last explanation only applies to banal things, to "common" objects. Any keen sensation, such *e.g.*, as crowd upon one in foreign travel, is at once received *as* new, not as remembered. We consciously apprehend it, and extend our mental horizon with no *blasé* sensation of previous experience. Does not the point lie in individual keenness of perception, or, on the other hand, as Vignoli insisted, in the power of anticipation, of imaginative construction, which from time to time coincides with the thing anticipated?

And in this aspect we may admit the imaginative or constructive element in dreams, as quite distinct from the dream illusions. Lalande excludes (*v.s.* Burnham) on the ground that the field of consciousness is so narrowed in dreams, as in insanity, that none of the usual landmarks of association by which we orientate ourselves can be present.

Modern psychology is coming more and more to recognise the existence of different types, or modes, of perception as of memory. (For the last, cf. *Les Crenels Calculateurs*. Binet, 1894.) And as our synthesis of temperament becomes more complete, the phenomena of paramnesia may prove as purely "constitutional" as chilblains or coloured hearing.

ARTICLES ON MEMORY.

Bourdon on Influence de l'age sur le mémoire immédiate. *Revue Philosophique*, August, 1894, gives statistics to show that "immediate" memory (which he correlates with attention and grasp of consciousness) increases from the age of 8 to 20, chiefly between 8 and 14, less from 14 to 20. He investigated 100 children, using series of 6, 7 and 8 figures or letters, and 5 and 6 words, and concludes that as a rule memory is in direct ratio with intelligence.

Ribot on Recherches sur le mémoire affective. *Ib.*, October, 1894, indicates a still unexplored region of memory, in which lie the images of olfaction, gustation, internal sensation, pleasure, pain and emotion in general. Can there be "spontaneous" revival of these images, or does their reproduction depend upon internal stimuli of the same character—an excited revival? Out of 60 experimental cases, 12 gave affirmative answers to spontaneous revival of all images of taste and smell, 48 could revive certain sensations only. Hunger, thirst, fatigue, pleasure and pain, were answered affirmatively. Emotions also, but in most cases this is true of the *intellectual* memory only; the true emotional state of feeling as well as circumstances is much rarer.

M. Ribot concludes that there is an "affective" type parallel to the "visual," "auditive," and motor types, consisting in the facile, complete, and preponderating revival of "affective" representations.

Individual differences of character are largely owing to it, while it proceeds rather from cerebral conditions ("*known or unknown*") than from intensity of primitive impression. To feel an emotion keenly, and to remember it keenly, are two distinct processes. The impression must be deep as well as vivid. Notably there is often an aggravation, or rolling up, a latent inculcation of painful memories, which relates to individual temperament. The article is merely thrown out to supplement the more adequate studies of "intellectual" memory already familiar to the psychologist.

Dugas on La Mémoire brute et la mémoire organisée. *Ib.*, November, 1894, points out that there are two essential types or "species" of memory analogous to the distinction between sensation and perception; besides the "varieties" visual, auditive, &c., more commonly insisted on. These two types he classifies as "rule" (*mémoire brute*), and organised (mémoire

organisée). The first is merely the passive repetition of a sensation, involuntary unconscious. The second involves interpretation and selection, conscious mental activity. Memory without effort is not co-ordinated; the mental process is one of ingestion, not digestion. The time sense is an important element in organised memory. The brain is master and not servant of its images. Reason and imagination enter into "true" memory; it is less a sum than a synthesis of images. And in diseases of memory dissolution of form is more fatal than "material" loss; it is not the number, but the nature and kind of facts obliterated that constitutes the gravity of amnesia.

SCHIFF'S "MEMOIRS."

Moritz Schiff on Gesammelte Beiträge zur Physiologie. (*Lausanne Benda*, 1894, Vols. i. and ii.)

Students of neuro-physiology will welcome this collection of Schiff's "Memoirs," of which the second volume has just been published under the editorship of Prof. Herzen. Besides a *précis* of his work on respiration and a number of early papers on vascular nerves, it contains several of his most important contributions to neuro-physiology. It is not a little remarkable to find that many of the vexed questions of the present day were attacked and answered by Schiff twenty or thirty years ago. Buried in some periodical publication they have been forgotten, and are only now rescued and reissued in a consecutive series to witness to the indomitable courage and perseverance of the veteran physiologist. Thirty years ago, before Darwin and others so familiar to us had written on the comparative physiology of plants and animals, Schiff (in an introductory lecture at Florence, 1863) proclaimed reflex action to be the distinguishing characteristic of animal life, evident even when the nervous centre producing it escapes observation. Another article on Cœnesthesia, published in 1871, is reprinted as a contribution to current theories of "consciousness," with special reference to the conclusions of Ribot (*Revue Philosophique*, November, 1892). Many of the papers are critical, and deal with the works of Du Bois-Reymond and Waller. Another series (1877-78) treats of inhibitory nerves. Schiff finds that stimulation of the nerve trunk produces inhibition of its peripheral branches in regard to certain stimulants—*e.g.*, the blood, in the case of cardiac inhibition. This effect he terms *negative irritation*, and describes as "a

particular kind of activity of the motor nerve suppressing a muscular contraction which would have been manifested if the nerve had not been irritated" (629). All Schiff's conclusions are supported by experiments, in regard to which he claims a value for the facts discovered, even where the theories leading to them may have to be rejected. (650.)

Vol. ii. treats of the physiology of muscle, cardiac innervation, and innervation of the lymph hearts.

A paper, dated 1858, on "The Direct Stimulation of Muscle," is republished with an appendix of 100 pp., dated 1894, which sums up the "idio-muscular," *v.s.*, "neuro-muscular" controversy, discusses the question whether the constant current, or its interruptions only, is a stimulus, and gives some excellent tracings.

The papers in the second section range from 1849 to 1882. In the third from 1850 to 1885.

As Schiff points out in the preface, the admirable arrangement and editing of these two volumes are due to M. Herzen. It was no easy task to bring such wealth of material, text and tracings into the consecutive form in which they have now been published, and the "Memoirs" will stand as a valuable and permanent contribution to physiology.

FRANCES A. WELBY.

Sensory Nerve of Muscles. (*Landauer.*)—In relation to the somewhat problematical "sense" commonly referred to as the muscular sense, proof of the existence or non-existence of sensory nerves in muscles is still extremely desirable, for the question cannot be considered as answered either by the much-quoted observations of Sachs, nor even by the more recent experiments of Chauveau. Nor, indeed, can they be considered as satisfyingly answered in this, the most recent investigation of the point. Landauer (who, by the way, commits two historical inaccuracies at the outset of his paper—the first attributing to Wundt the opinion that the muscular sense is of central origin, and the second attributing to Bell the discovery that is generally associated with his name) examined frog's muscle by various methods, and says that, besides broad motor nerve-fibres, he found much finer nerve-fibres to all appearance sensory and vaso-motor. He comes to the conclusion from finding that subsequent to section of the anterior roots the motor nerves undergo de-

generation while the sensory do not. He seems to think that ordinary motor fibres give off finer branches that are vaso-motor. He gives figures of his presumably sensory nerve-endings, but does not really give much proof that muscle, as distinguished from the connective tissue of muscle, possesses sensory nerve-ending.

A. D. W.

Langendorff (*Pflüger's Arch.*, 1894, p. 521) finds that the surprisingly rapid loss of irido-constrictor power of the motor oculi *post-mortem* is due to the intervening ciliary ganglion. Excitation of the third nerve above the ganglion quickly ceases to produce any effect. Excitation of the third nerve below the ganglion has a powerful effect long after death, *viâ* the short ciliary nerves. Constriction of the pupil *post-mortem* is conveniently demonstrated on the rabbit's eye by laying the optic stump across the electrodes; this excites the long ciliary (dilatators) as well as the short ciliary (constrictors), but constriction predominates. The ciliary ganglion, then, is a ganglion traversed by *motor* fibres, and not an analogue of posterior root ganglia.

A. D. W.

Betheiligung der Nerven an der Schwankungen in der Pupillen Wiete. *Pflüger's Archiv.* June 1, 1894.)

Excitation of the cervical sympathetic causes, in addition to the well-known dilatation of the pupil on the same side, reflex constriction of the pupil on the opposite side, presumably due to retinal excitation of the eye whose pupil is dilated. Excitation of the central end of the vagus gives reflex constriction of the pupil on the same side, reflex dilatation on the opposite side. These effects vary with the state of excitation of the animal (cat, rabbit).

A. D. W.

Wundt on Psycho-Physical Interactions.

In a recent number of *Philosophische Studien* (x. 1, *Ueber psychische Causalität, und das Princip des psychophysischen Parallelismus*), Wundt points out that psycho-physical interactions are two-fold: physico-psychical (centripetal) when the

external stimulus produces a psychical result in the organism, resulting in *sensation;* psycho-physical (centrifugal), when the psychical process, *e.g.*, will, motives, has a physical effect and produces *movement*. There is thus an empirical basis for psycho-physics, but it is needful to protest against the current tendency to conclude that this covers the whole field of psychology. For example, the parallelism can only apply to processes of consciousness. The physical concomitants of *unconscious* psychical processes can only belong to metaphysics, and again no association of physical processes can explain the nature of the connection between psychical elements any more than our ideas are able to throw light on the physiological processes which accompany them. Wundt criticises sharply the "materialistic psychology" of Münsterberg and Ziehen. These psychologists, he says, throw "matter" out of the window as unthinkable, and bring it in again under the name of "material processes." They affirm matter to be a hypothesis, and yet use it to explain psychological phenomena. The old materialism at least believed in "matter" *per se* and in its molecular motions. Here we have really a new metaphysic, the more dangerous in that it is sheltered behind the theorems of natural science. Experimental psychology, moreover, reduces psychological processes to anatomical elements, until it seems almost superfluous to maintain with Münsterberg that psychology *is* a science. It rather appears as an appendix to muscular contraction. The real subject of psychology is not the physical but the psycho-physical individual. "Bad analogies," concludes Wundt, "are intellectual opiates," and the soul is not explained, though it may be interpreted, by reflex determinism. The rest of the number is occupied by articles on *das Massprincip der psycho-physik*, by F. Lange; *die Abhängigkeit-zwischen Reiz u. Empfindung*, Merkel, part iv., and Dr. Van Biervliet's account of experiments to show that the duration of reaction-times is considerably affected by the pulse-rate.

II. *Die Empfindung als Funktion der Reizænderung. Ebbinghaus's Zeitschrift für Psychologie u. Physiologie der Sinnesorgane* (vii., 4). Preyer extends Du Bois-Reymond's law of muscular contraction in response to electrical excitation, to all sensory and motor nerves, in response to any adequate stimulus. He finds that the threshold of sensation may be considerably raised with a continuous and steadily increasing stimulus, whereas sudden changes or interrupted stimuli produce sensations at a lower level. The sensation or the contraction corresponds, not to the actual magnitude of the stimulus, but to its alteration from

moment to moment, and is also determined by the rapidity and time-duration of the stimuli. The nervous system has in its evolution learned to respond to marked and rapid changes of excitation, rather than to small or steady stimuli. In a former number of the *Zeitschrift* (vi. 472) Scripture had given applications of the same law to changes of sound and of temperature. A very gradual change of stimulus may raise the pitch as much as a whole tone imperceptibly, whereas more rapid changes produce an immediate change of sensation. In temperature changes, also, a gradual rise of 10° C. or more may be unnoticed, although a sudden change of $\frac{1}{4}$° is perceptible. The unnoticed pressure of the atmosphere is another instance. In *Zwei Fälle von Grunschen* (*ib.*, p. 305), Somya draws attention to the rare phenomenon of chloropia or green vision. He attributes it to minute changes in the choroid, and not, as has been supposed, to retinal modification.

In regard to the question of central, *versus* peripheral, fusion of sense-impressions in the case of sound, Wundt (*Phil. Stud.*, viii., 641, 1893, *Ist der Hör-Nerv direct durch Tonschwebungen erregbar*) sums up the arguments of Scripture and Karl Schäfer, as well as the earlier work of Cross and Goodwin, and Sylvanus Thompson. The conclusions of Scripture (and also of Ewald) run counter to the theory of specific nervous energies, according to which each auditory fibre can convey one tone only, whatever the nature of the stimulus. Wundt refers to other analogous phenomena equally unfavourable to this theory, *e.g.*, the beats and beat-tones of König, which indicate that beats can arise in the brain without interference at the periphery, and that direct periodical stimulation of the auditory nerve can, at a certain frequency, give rise to tone-sensations. Even if we accept the theory of Helmholtz, it connotes a mechanical excitation of the nerve-fibres at the organ of Corti; and it is equally conceivable that direct excitation of the nerve might occur without peripheral intervention. Ewald, in his investigations on the end organs of the auditory nerve, found that pigeons whose labyrinths had been extirpated still reacted to auditory stimuli, whence he concluded that the sound waves directly stimulated the stump of the nerve by transmission through the bones of the cranium. A later series of observations (*Die centrale Entstehung von Schwebungen zweier monotisch gehörten Töne, Pflüger's Archiv.*, June 1, 1894) seem to justify the same conclusion in regard to man. It would follow that when deaf people hear sounds conducted through the cranial bones this is not a proof, as is generally supposed, of the

integrity of the labyrinth. If the trunk of the nerve is capable of direct stimulation, the labyrinth might in these cases be completely disorganised. Again, if the nerve be thus directly excitable, the case for a central origin of beats is greatly strengthened, seeing that interference in the terminal apparatus is not an indispensable condition of their production.

K. Schäfer maintains that the bones of the cranium conduct sound from one ear to the other, thus making peripheral fusion. Ewald, however, arranged two tuning-forks so that they could each be heard by one ear only. One was tuned to give 130 vibrations per second, the other to a slightly higher rate. Heard separately there was no indication of oscillation; heard together there was a distinct and easily-counted beat. The conclusion is that sounds conveyed separately to each ear may be *centrally* fused into a beat. The beat moreover coincided with subsequent binaural observations with the same tuning forks. The case seems to stand upon the same footing as the analogous case of the eye, in which the balance of evidence is to the effect that two complementary colours presented separately to each eye may give rise to the sensation of a single resultant colour, the mechanism of which fusion must obviously be central.

Another interesting analogy between eye and ear is that established by Zwaardemaker (*Das presbyacousische Gesetz Zeitschrift für Ohren-heilkunde*. June, 1893. *Der Umfang des Gehörs in den verschiedenen Lebensjahren. Ebbinghaus Zeitschrift*. March, 1894) who formulates a law of what he terms presbyacousia. He gives a curve founded on 219 observations to show that the range of hearing of the upper notes diminishes from *e* 7 (11,440) max. in infants, to *a* 6 (28,160) or even *g* 6 (25,344) in adults. He refers to an earlier paper in proof of the same limitation in regard to the lower notes, which, however, he admits to be far more uncertain. In short, from the age of 4 to 70, hearing, he says, undergoes a continuous restriction, losing as much as half an octave. In the second paper he alludes to the inductively established fact that bone conductivity decreases with age. He connects the beginning of the decline curve of hearing with the end of bone-growth.

Frances A. Welby.

Association by Unconscious Middle Factors. Jerusalem, Wundt.—*Phil. Stud.*, x., ii.

Dr. Jerusalem communicates an experience of Herr Baumgarten, purporting to show that the idea or memory-image of two people, vividly called up by the unnoticed scent of a flower (which was not detected till after the memory-image had arisen in consciousness) implies that "the smell-sensation was here a really unconscious middle factor." He adduces this in support of Scripture's views (*Phil. Stud.*, vii.).

Wundt rejoins that the scent of the flower was "unrecognised" (*unbemerkt*), not unconscious (*unbewusst*). Baumgarten's observation is interesting, because the unrecognised middle factor is here present in consciousness, whereas in Scripture's experiments the middle factors were not direct sensations during the process of mediate association, but were reproduced (vii., p. 78). Here the scent was *perceived*, though not *apperceived*, *i.e.*, not distinguished from the scent of the other flowers. Wundt, therefore, calls this "a case of mediate association through middle factors present in consciousness, but only dimly perceived there."

FRANCES A. WELBY.

Dana and Ketscher on Paralysis Agitans.

I.—In some preliminary remarks on the clinical phenomena of the disease, Dana (*New York Medical Journal*, June 10, 1893), emphasizes three classes of symptoms.

(1) Motor symptoms, chiefly due to difficulties in commencement of movements, or, as the author terms it, "a hitch in volition."

(2) Vasomotor symptoms, flushings, sweats, subjective sensation of heat, peculiarities of pulse. He believes there is a general vasomotor paralysis, affecting the vessels of the cord and the nerve trunks, in addition to those of the skin.

(3) Disturbances of the blood, and of metabolism. Purpura hæmorrhagica occurs sometimes, glycosuria sometimes; and in the opinion of some authors, phosphaturia. The author examined the blood in two cases; in one, the chief abnormality was absence of eosino-philous cells; in the other, there was "anæmia of chlorotic type; lessened percentage of hæmoglobin, and decreased resistance of red cells."

(4) Sensory symptoms, viz., sciatica, neuralgia, and rheumatic pains; distressing sensations of heat and cold.

He then turns to the pathology of the disease with which his paper is chiefly occupied. He quotes 44 cases of paralysis agitans, real or supposed, in which *post-mortems* have been made, but out of these he rejects all but fourteen, as being in his opinion scientifically valueless. Upon the cases of Dubief, von Sass, Koller, and Borgherini, he places most stress. In the bulk of these cases certain anatomical facts appear, which although rather vague may possibly be taken as the basis of the morbid anatomy of this disease. They are degeneration of the vessels, enlargement of the perivascular spaces, proliferation of the neuroglia and connective tissue in the neighbourhood of the vessels; pigmentation and some degeneration of the spinal motor cells, and of those in the bulbar nuclei; thickening of the pia mater surrounding the cord, with sclerosis of the neighbouring periphery of the cord; blocking of the central spinal canal by proliferation of cells; changes in the cells of the sympathetic.

The author's own case is as follows:—A woman, age about 58 at time of death, died from exhaustion five years after onset of the disease. The symptoms began after a fright, and consisted of pain and weakness in the back; tremors of the limbs, followed by weakness; stooping gait; slowness of movement, &c. The brain and cord were hardened in Müller's fluid. In the cortex cerebri was some increased vascularity, dilatation of the vascular spaces; and some trivial changes in the nerve cells.

In the nuclei of the seventh, ninth, tenth, and eleventh cranial nerves was pigmentation, with some degeneration of the cells.

In the cord, the pia mater, particularly of the cervical and lumbar regions, was thickened; while in the lateral columns beneath was some diffuse infiltration of connective tissue, which had not however caused much degeneration of the nerve fibres. The cerebellar tracts were not affected by this sclerosis. The vessels in the anterior cornua were much dilated, particularly the veins. The anterior cornual cells were somewhat shrunken and pigmented, and took stains irregularly. The central canal, at some levels, was filled with *débris* and exsudate. The anterior nerve roots were congested, and perforated with dilated vessels.

In a second case, the details of which are not fully given, the author found marked thickening of the spinal pia mater: with diffuse interstitial sclerosis, of the lateral columns especially; congestion of the central and anterior gray matter; and slight changes in the nerve cells.

The anatomical seat of the disease the author places in the spinal cord, medulla and pons, and more particularly in the blood vessels of the central and anterior gray matter of the cord, and in the lateral columns (pyramidal tracts, lateral fundamental columns, and lateral limiting layers). As to the nature of the anatomical change we quote his own words:—"This anatomical basis I believe to be essentially as follows: First, a congestion of the central and anterior parts of the gray matter of the cord with some thickening of the blood vessels, but not very much, and some changes of the external wall of the nature of periarteritis. With this congestion and vascular irritation there occurs a proliferation of connective tissue, which especially invades the lateral columns of the spinal cord. In severe cases it affects also the periphery, producing a leptomeningitis and a zone of selerosis surrounding the cord in a variable degree. The congestion with dilatation of blood vessels affects especially also the anterior roots, to a less extent the posterior roots. There is degeneration of the cells of the anterior horns, which, however, occurs only late in the disease and does not affect all the cell groups. The central cells or the cells of the median and central areas, which we suppose to be more connected with vasomotor and glandular functions, seem to be especially involved. There is also—and this is an important fact to bear in mind—a decided loss of the fibre net-work in the anterior horns and the central gray matter. The changes appear to involve especially the cervical and lumbar regions. Similar changes occur in the medulla and pons, but here they are less marked. Degenerative changes, however, are to be found, in many cases at least, in the cells of the nuclei of the glosso-pharyngeal and pneumo-gastric nerves. Such changes would explain the disturbances in the vasomotor function and heart action found in the disease."

He makes this further suggestion as to the causation of the disease, viz., that it is due to the presence, in the circulation, of some irritative toxine, which in its turn may originate in some glandular defect—the process being analogous to that of myxœdema. For this hypothesis, however, no evidence is adduced.

II.—Ketscher's paper (*Zeitschrift für Heilkunde*, 1892, p. 445) opens with a very complete account of the work already done upon the morbid anatomy of paralysis agitans. It would appear that, after dismissing those cases which probably were not genuine paralysis agitans at all, the remainder consist partly of cases in which no morbid appearances were discovered *post-*

mortem (some of which, however, were not examined microscopically), and partly of others in which more or less definite results were obtained. These results may be summarised as—hyperplasia of the connective tissue and of the neuroglia in the spinal cord; changes of various degrees in the vessels, pigmentation of the ganglion cells, blocking of the central canal of the spinal cord, presence of corpora amylacea in more or less excess. Similar alterations have also been found in the brain, the peripheral nerves and muscles, and in the sympathetic system. Most investigators consider that the chief seat of the disease is in the spinal cord; though they are by no means unanimous on this point; and some distinguished men (Charcot, Oppenheim, &c.) maintain that paralysis agitans is a "pure neurosis."

Ketscher investigated microscopically three cases of paralysis agitans; the ages of the patients were respectively 70, 68 and 76. In all three cases he found changes in the central nervous system (mainly, it would appear, in the cord); in two cases changes in the muscles, in one case in the peripheral nerves. These changes he summarized as follows:—

(1) As to the parenchyma.

Scattered indication of degeneration and atrophy, viz., in the ganglion cells of brain and cord—pigmentation, indistinctness of outline, absence of nuclei, finely granular degeneration; in the nerve fibres (particularly of the posterior columns of the cord)—changes ranging from mere swelling of the axis-cylinder to complete disappearance of the nerve fibres; in the muscles—multiplication of nuclei, loss of transverse striation, hyaline and fatty degeneration, disappearance of muscle-substance.

(2) As to the interstitial tissues.

There was general increase of the interstitial connective tissue. Around the cord the peripheral connective tissue was thickened, and broad bands extended from it towards the central parts, particularly in the posterior columns. In the peripheral nerves, the peri-neurium was thickened, and in some places the endo-neurium also. Similarly the connective tissue between the muscular fibres was thickened.

The neuroglia both of brain and cord was increased. The neuroglia layer of the cerebral cortex, and the ependyma of the ventricles was thickened. So was the glia of the cord, both the peripheral layer and that of the internal parts, and this overgrowth appeared to start from the neighbourhood of the vessels. In the posterior columns particularly this overgrowth penetrated

not only between groups of fibres, which were thus marked off into separate bundles, but also between the individual nerve fibres in some places. The disease reached its maximum in the lumbar region of the cord. There were numerous corpora amylacea, in the ependyma of the lateral ventricles, in the superficial parts of the brain, in the peripheral parts of the cord, and along the posterior fissure of the cord.

The central canal of the cord was blocked with proliferated epithelial cells.

(3) As to the vascular system.

The vascular walls were thickened. Here and there were miliary aneurysms, or even small extravasations of blood. The adventitia of the cerebral vessels contained a good deal of pigment. The lymphatic spaces—peri-vascular and peri-cellular —were dilated, and contained sometimes coagulated lymph, sometimes leucocytes, sometimes blood. Here and there in the neighbourhood of these spaces an "œdematous softening" of the brain substance could be made out.

These changes, which have been observed by others besides himself, the author believes to be no fortuitous occurrence, but to stand in some causal relation to the disease.

A certain reservation has, however, to be made in view of the author's investigations into the condition of the nervous system in aged people generally. He made microscopical examination of the nervous system in ten aged people, dying from various causes; and comes to the conclusion that in old age changes are found resembling in kind those found in paralysis agitans, and differing only in their less advanced degree. He finally expresses his agreement with the view of Jacobsohn, Dubief and Borgherini, that paralysis agitans is nothing else than senility of the nervous system, early in occurrence and intense in degree.

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